REPORTS

OF THE

SOCIETY FOR THE STUDY OF DISEASE IN CHILDREN.

VOLUME V.

Session of 1904–1905.

EDITOR:

GEORGE CARPENTER, M.D.

Hon. Secretaries:

LONDON:

EDMUND CAUTLEY, M.D. FRANCIS JAFFREY.

PROVINCIAL:

R. H. ANGLIN WHITELOCKE, C.M. (Oxford).

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OFFICE BEARERS AND MEMBERS OF THE COUNCIL FOR THE SESSION OF 1905-1906.

ELECTED AT THE ANNUAL GENERAL MEETING.

Council (1905-1906).

Those marked thus (*) served on the Council or held office during last session, 1904–5.

JOSEPH BLUMFELD, M.D.
*ROBERT CAMPBELL, M.B. (Belfast).
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*PERCY LEWIS, M.D. (Folkestone .
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*A. H. TUBBY, M.S.
*J. THOMSON WALKER, C.M.
F. PARKES WEBER, M.D.

Honorary Treasurer.

*R. CLEMENT LUCAS, B.S.

Honorary Editor of Reports.

*GEORGE CARPENTER, M.D.

Honorary Secretaries.

LONDON:

*EDMUND CAUTLEY, M.D. FRANCIS JAFFREY.

PROVINCIAL:

*R. H. A. WHITELOCKE, C.M. (Oxford).

LIST OF OFFICE-BEARERS SINCE THE FORMATION OF THE SOCIETY IN THE YEAR 1900-1901.

Dates.	Chairmen of Council.	Honorary Secretaries.		Honorary Treasurer.	Editor of the 'Reports.'
		London.	Provincial.		
1900–1	A. E. Sansom, M.D.	Sydney Stephenson, C.M.	Charles H. Willey, C.M. (Sheffield).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1901–2	A. E. Sansom, M.D.	Sydney Stephenson, C.M. George Carpenter, M.D.	Theodore Fisher, M.D. (Bristol).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1902–3	A. H. Tubby, M.S.	Sydney Stephenson, C.M. George Carpenter, M.D.	Theodore Fisher, M.D. (Bristol).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1903–4	Sydney Stephenson, C.M.	George Carpenter, M.D. G. A. Sutherland, M.D.	Theodore Fisher, M.D. (Bristol).	R. Clement Lucas, B.S.	George Carpenter, M.D.
1904–5	Sydney Stephenson, C.M.	G. A. Sutherland, M.D. Edmund Cautley, M.D.	R. H. A. Whitelocke, C.M. (Oxford).	R. Clement Lucas, B.S.	George Carpenter, M.D.

RULES

Name.

1. The name of the Society shall be "The Society for the Study of Disease in Children."

Object of the Society.

2. The object of the Society shall be the study of disease and of allied subjects in children.

Constitution.

3. The Society shall consist of Ordinary, Temporary, and Honorary Members.

Qualification for Membership.

4. All duly registered medical men shall be eligible for membership.

Officers.

5. The officers of the Society shall be elected from the ordinary members, and shall include an Honorary Treasurer, an Honorary Editor of 'Reports,' Honorary Secretaries (one of whom shall be resident in the London postal district, and one in some other part of Great Britain or Ireland), and twenty-four other members, of whom at least one third shall not be resident in the London postal district. These together shall constitute the Council and manage the affairs of the Society.

Ordinary Members.

6. The names of candidates shall be proposed upon a printed form provided for the purpose, and signed by two or more members from personal knowledge. The names of candidates shall in the first instance be submitted to the Council, in which shall be vested the absolute power of declining to allow the

nomination to proceed. The proposal paper, after approval by the Council, shall be read at an Ordinary Meeting of the Society; and the ballot shall take place at the next meeting. No election shall be valid unless at least ten members vote, and no candidate shall be elected who fails to obtain four fifths of the votes recorded. No rejected candidate shall be eligible for proposal until twelve months have elapsed.

Form of Admission.

7. Every Ordinary Member, after his election, and after payment of the annual subscription, and of the entrance fee (if any), shall sign the roll of members, declaring thereby his adhesion to the Rules of the Society.

Temporary Members.

8. Any resident medical officer of a hospital or infirmary shall be eligible for election by the Council as a Temporary Member of the Society for a period of twelve months, without payment of any subscription or entrance fee. He shall be entitled to attend the ordinary Meetings, but he shall not have a vote, shall not be eligible for office, shall not be entitled to receive a copy of the 'Reports,' and shall not introduce visitors. If, at the termination of the period for which he has been elected a Temporary Member, he desires to become an Ordinary Member of the Society, he must go through the usual form of election, but shall be exempt from paying any entrance fee.

Honorary Members.

9. The Council shall have power to propose for election as Honorary Members the names of persons eminent in medicine, surgery, or the collateral sciences. The Honorary Members, who shall not exceed twelve in number, shall be balloted for, after proposal by the Council, at a General Meeting of the Society, and be subject to the same rules as govern the election of ordinary members. They shall enjoy all the privileges of ordinary members, voting excepted. They shall not be called upon to pay any fee or subscription.

Resignation and Expulsion of Members.

10. Any member may retire from the Society on giving written notice of the intention to do so to one of the Secretaries and on paying any contributions that may be due.

The name of any member may be expunged from the Society's roll only at a General Meeting convened by the Council specially

for consideration of the case. At least ten votes must be recorded, and four fifths of the votes, if recorded in favour of the expulsion, shall be decisive. Any person expelled in accordance with this rule shall forfeit all claim upon the Society.

Subscriptions.

11. The annual subscription for Ordinary Members shall be One Guinea, payable in advance at the beginning of the session in October. Ordinary Members elected after July the 31st, 1901, shall pay an entrance fee of One Guinea; but in the case of any member elected subsequently to Easter in any year the member shall not be called upon to pay a subscription during the ensuing session. Original Members pay no entrance fee. A member shall at any time be exempted from further annual subscriptions by paying a composition fee of Ten Guineas.

A member whose subscription is six months in arrear shall be reminded of the fact by one of the Honorary Secretaries; and if the amount due remain unpaid during the current year, his name may be removed by the Council from the list of members.

Election of Officers.

12. The Officers of the Society shall be elected by ballot at the Annual Meeting. No member, except the Honorary Treasurer, may hold the same office for a longer period than three consecutive years. A list of names recommended by the Council for election shall be sent to each ordinary member at least fourteen days before the Annual Meeting of the Society. A name or names may be substituted on the voting paper for that of any member or members proposed by the Council for election. Two Scrutineers shall be appointed by the Chairman at the commencement of the Annual Meeting, and their duty shall be to receive and examine the balloting lists, and report the results to the meeting. In the case of equal voting the Chairman shall decide by lot.

Honorary Treasurer.

13. The Honorary Treasurer shall have charge of the funds of the Society, and keep an account in an orderly and business-like way of all sums that pass through his hands. He shall receive all moneys due to, and pay all moneys due from, the Society, only after the accounts have been passed by the Council and signed by two of the members present. He shall keep a printed receipt-book for subscriptions and fees, and every receipt shall be signed by him and countersigned by one of the Honorary Secretaries. He shall prepare an annual statement of accounts, which shall be audited by two ordinary

members of the Society not in office, and presented, with the Council's Report, to the next Annual Meeting. He shall keep a separate banking account in the name of the Society, and shall invest the funds from time to time as directed by the Council. The Honorary Treasurer shall be *ex officio* a member of Council.

Honorary Editor of 'Reports.'

13A. The Honorary Editor of the 'Reports' shall edit the Society's 'Reports' and he shall keep all papers and communications and illustrations intended for publication in the 'Reports.' He shall be *ex-officio* a member of the Council.

Honorary Secretaries.

14. The Honorary Secretaries (or one of them) shall conduct all correspondence, notify to new members their election, and convene, attend, and minute all meetings of the Society, of the Council, and of any committees appointed by the Council. They shall arrange the order of proceedings at all meetings. They shall submit to the Council all official communications and all nominations for membership in the Society. They shall keep a register of all cases and papers communicated to the Society, and, unless otherwise decided by the Council, shall act as editors of all publications authorised by the Council. They shall keep a roll of the officers and members of the Society. In the discharge of these various duties the Secretaries shall have power to employ clerical assistance, to use printed or typed forms, and to prepay postages and telegrams. They shall be *ex officio* members of Council.

Meetings.

15. These shall be *Ordinary*, *Special*, and *General*.

Ordinary Meetings shall be held at hospitals in London or elsewhere, or in places appointed by the Council. There shall not be more than eight Ordinary Meetings in the year, and of these one at least shall be held elsewhere than in London. The meetings shall take place upon the third Friday in the months of October, November, January, February, March, April, May, and June, at 5.30 p.m., unless otherwise arranged by the Council. They shall not be of longer duration than two hours, except by special resolution of the Meeting. Clinical and Pathological Demonstrations, and discussions thereon, shall take precedence over papers. Nothing relating to the laws or the management of the Society shall be discussed at an Ordinary Meeting.

Special Meetings may be convened by the Council for the purpose of discussing a subject or subjects deemed to be of

particular interest in relation to disease in children, or to allied subjects.

A *General Meeting* shall be called by the Honorary Secretaries on the requisition of any five members of Council or of any twenty ordinary members of the Society. At least fourteen days' notice shall be given to every ordinary member of the Society; and the character of the business to be transacted at such meeting shall be stated on the summons paper. No business other than that set forth upon the summons paper shall be considered at the meeting.

The *Annual General Meeting* shall be held at a time and in a place determined by the Council, fourteen days' notice having been given to every ordinary member of the Society. Its business shall include election of officers, presentation of the annual Report of the Council, and the discussion of any proposed alteration or alterations in the rules (notice of which shall be given in the summons convening the meeting). Ten ordinary members shall form a quorum. No change in the rules shall be valid unless three fourths of the votes are recorded in favour of the alteration. A notice, written or printed, may be served upon any member, either personally or by sending it through the post in a prepaid letter, addressed to such member at his registered address.

Chairman.

16. A Chairman, who must be an ordinary member of the Society, shall be chosen by show of hands or by ballot at the commencement of each meeting, whether ordinary, special, or general. He shall sign the minutes of the preceding meeting or meetings, regulate the proceedings, check irregularities, interpret the application of the laws, and enforce the recognised rules of debate. The Chairman shall have an ordinary and a casting vote.

Publications.

17. Reports shall be published in such manner and at such times as the Council may direct. A copy shall be forwarded free to every Honorary Member of the Society, and to every ordinary member whose subscription is not in arrear.

Visitors.

18. An ordinary member may introduce two medical men as visitors at ordinary meetings of the Society. Visitors shall sign their names in the book provided for the purpose. On the invitation of the Chairman, visitors shall be permitted to take

part in the discussions of the Society. The same visitor shall not be introduced more than twice during one session.

Council.

19. The Council shall have the management of the affairs of the Society, and shall meet as often as may be necessary for the transaction of business. Seven days' notice of each meeting shall be sent by the Honorary Secretaries to every member of Council. Five members shall form a quorum. The Council shall determine questions, if necessary, by ballot. The Council, at its first meeting, shall elect from amongst its members a Chairman, to preside over its deliberations during the session. He shall not hold office for longer than two consecutive years. In his absence the chair shall be taken by some other member of Council, duly proposed and seconded. The Chairman of Council shall have a casting, in addition to an ordinary vote. The Council shall from time to time make regulations, issue instructions, appoint committees, and take any other lawful steps to further the good government of the Society. It shall prepare and present to the Annual Meeting a Report upon the state of the Society. The Council shall be empowered to fill all vacancies that may occur from death or otherwise between one Annual Meeting and another. It shall decide all questions relating to the publication of the 'Reports.' It shall annually depute three of its members, with the Honorary Secretaries, to form a *Committee of Reference*, the duty of which shall be to determine the acceptance of communications referred to it by the Honorary Secretaries. The Council shall be elected by ballot at the Annual Meeting. One third of its members, exclusive of the Honorary Treasurer and the Honorary Secretaries, shall be replaced annually by an equal number of ordinary members of the Society. An Extraordinary Meeting of Council may be called (1) by the Chairman of Council, or (2) by the Honorary Secretaries, and (3) shall be called by the Honorary Secretaries on the requisition of any five members of Council. Seven days' notice of such extraordinary meeting must be given in writing by the Honorary Secretaries to every member of Council upon a summons paper specifying the object for which the meeting is called.

REPORT OF THE COUNCIL, 1904–5.

Read at the Annual General Meeting, held at No. 11, *Chandos Street, Cavendish Square, W., on Wednesday, July the* 19*th,* 1905.

The Council have pleasure in reporting that the total membership of the Society is now 332, there having been 48 new names added to the roll during the current session. They have accepted with great regret the resignation of Dr. W. A. Wills, owing to ill-health, and desire to express their appreciation of his services to the Society and to the Council. The Society has also to mourn the loss by death of Mr. Herbert Allingham.

The Ordinary Meetings of the Society continue to be well attended, and many interesting cases and specimens have been demonstrated and discussed during the session. A Special Meeting was held in December for the purpose of a discussion on "Sudden and Unexpected Death in Children." The medical aspects of the subject were dealt with by Dr. C. J. Macalister, Dr. J. Porter Parkinson, and Mr. J. W. Thomson Walker, while Mr. A. H. Tubby discussed the surgical causes, and Dr. J. Blumfeld death during anæsthesia. The Wightman Lecture was delivered in May by Dr. Henry Ashby, who took as his subject, "Some of the Neuroses of Early Life." The lecture was very much appreciated by all who were present, and a very cordial vote of thanks was accorded to the lecturer on the motion of Dr. William Ewart, seconded by Dr. Leonard Guthrie.

The Provincial Meeting was held at Sheffield on Saturday, June the 17th. A large number of interesting clinical cases and pathological specimens were demon-

strated at the Children's Hospital, and the meeting then proceeded to the reading and discussion of papers. In the evening the members and their friends dined together, under the chairmanship of Dr. C. N. Gwynne. The Meeting was a most successful one, and the Council desire to express their indebtedness to Dr. Gwynne and Dr. Willey, who kindly undertook the necessary local arrangements, and to the members and non-members of Sheffield who supplied clinical cases and papers.

The Council have invited Dr. Broca, President of the Society of Pædiatrics of Paris, to deliver the Wightman Lecture in 1906, and are pleased to report that he has kindly consented to do so.

The finances of the Society are in a satisfactory condition, as will be seen from the Treasurer's report.

TREASURER'S REPORT.

The Treasurer has much pleasure in drawing attention to the sound financial position of the Society.

A balance of £149 9*s*. 10*d*. was carried forward from last year's account, allowing a further investment of £100 in Consols, thus bringing the invested funds up to £341 10*s*. 2*d*. in 2½ per cent. Consols. The subscriptions collected amounted to £347 11*s*. 0*d*., which, together with the balance sale of Reports and dividends, brought the available assets up to £514 3*s*. 7*d*. Of this sum £307 19*s*. 6*d*. have been expended, leaving a balance to be carried forward of £206 4*s*. 2*d*. There was a balance last year of £20 for the Wightman Lecture of this year, which was drawn in favour of Dr. H. Ashby, and a further subscription of £20 has been received from Mr. Wightman and remains in hand for the Wightman Lecture of 1906.

R. CLEMENT LUCAS.

BALANCE SHEET FOR THE YEAR ENDING JULY, 1905.

Receipts.	£	s.	d.
To Balance brought forward	149	9	10
,, Subscriptions	347	11	0
,, Messrs. Churchill, Sale of Reports ...	9	13	10
,, Dividends on Consols	7	8	11
	£514	3	7

Payments.	£	s.	d.
By Hollebone Bros. & Trench, purchase of £112 16*s.* 8*d.* 2½ % Consols, making £341 10*s.* 2*d* invested	100	0	0
,, W. Straker, Limited	1	8	2
,, G. Pulman and Sons (£13 15*s.* 3*d.* and £5 9*s.* 8*d.*)	19	4	11
,, Adlard and Son	143	6	4
,, Medical Society of London	18	18	0
,, Dr. George A. Sutherland	1	15	0
,, Mr. H. Dickinson	20	7	0
,, Treasurer's Petty Cash	3	0	0
	307	19	5
,, Balance carried forward	206	4	2
	£514	3	7

INVESTMENTS.

Consolidated Stock, 2½ %	£341	10	2

WIGHTMAN LECTURE FUND.

Balance brought forward	£20	0	0	Paid Dr. Ashby	£20	0	0
Per Mr. Wightman	20	0	0	Balance in hand	20	0	0
	£40	0	0		£40	0	0

We, the undersigned, have examined the foregoing accounts, together with the Vouchers and Bank Book, and find them to be correct.

FREDERIC F. BURGHARD,
H. S. CLOGG, } *Auditors.*

July 17th, 1905.

Meetings of the Society were held during the Session of 1904–1905 *at the undermentioned places.*

1. Friday, October the 21st, 1904.—No. 11, Chandos Street, Cavendish Square, W. Dr. Fletcher Beach in the Chair.
2. Friday, November the 18th, 1904.—No. 11, Chandos Street, Cavendish Square, W. Mr. Robert Jones (Liverpool) in the Chair.
3. Friday, December the 16th, 1904 (Special Meeting for the discussion of "Sudden and Unexpected Death in Children").—No. 11, Chandos Street, Cavendish Square, W. Dr. Frederick Taylor in the Chair.
4. Friday, January the 29th, 1905.—No. 11, Chandos Street, Cavendish Square, W. Dr. Percy Lewis (Folkestone) in the Chair.
5. Friday, February the 17th, 1905.—No. 11, Chandos Street, Cavendish Square, W. Dr. Edmund Hobhouse (Hove) in the Chair.
6. Friday, March the 17th, 1905.—No. 11, Chandos Street, Cavendish Square, W. Dr. Leonard Guthrie in the Chair.
7. Friday, April the 14th, 1905.—No. 11, Chandos Street, Cavendish Square, W. Mr. R. Clement Lucas in the Chair.
8. Friday, May the 5th, 1905.—No. 11, Chandos Street, Cavendish Square, W. Dr. George Carpenter in the Chair.
9. Friday, May the 19th, 1905 (the Wightman Lecture on "Some of the Neuroses of Early Life," by Dr. Henry Ashby, Manchester).—No. 11, Chandos Street, Cavendish Square, W. Mr. R. Clement Lucas in the Chair.
10. Saturday, June the 17th, 1905 (Provincial Meeting).—The Children's Hospital, Western Bank, Sheffield. Dr. George Willey (Sheffield) in the Chair.
11. Wednesday, July the 19th, 1905 (Annual General Meeting).—No. 11, Chandos Street, Cavendish Square, W. Dr. Robert Hutchison in the Chair.

LIST OF OFFICE BEARERS, PAST MEMBERS OF COUNCIL, WITH DATES OF SERVICE, AND MEMBERS OF THE SOCIETY FOR THE SESSION 1905—1906.

(*Revised October*, 1905.)

(O.M. = Original Member. T. = Treasurer. S. = Secretary. S with date attached = Past Secretary. P.S. = Provincial Secretary. P.S. with date attached = Past Provincial Secretary. Ed. = Editor of the Reports. C. = Member of the Council. C with date attached = Past Member of Council. * = Life Member. C.C. = Chairman of Council. C.C. with date attached = Past Chairman of Council. W = Wightman Lecturer. W with date attached = Past Wightman Lecturer.

Elected

O.M. Abrahams, Bertram Louis, M.B., Assistant-Physician at Westminster Hospital; 14, Welbeck Street, W.

O.M. Achard, Alexander Louis, M.D., 34a, Gloucester Place, W.

1902 Adams, Charles E., 227, Gipsy Road, S.E.

O.M. Alliott, Alexander John, M.D., Surgeon at The Hip Hospital, Sevenoaks; The Vine, Sevenoaks.

1904 Anderson, C. M., M.D., 146, Gloucester Terrace, Hyde Park, W.

1903 Anderson, J. Ford, M.D., 41, Belsize Park, N.W. (C. 1903–4, 1904–5.)

1901 Archdale, Mervyn A., M.B., East Riding Lunatic Asylum, Beverley.

1902 Armour, Donald John, M.B., Assistant-Surgeon at The West-London Hospital; Senior Assistant-Surgeon at The Belgrave Hospital for Children; 89, Harley Street, W.

Elected

O.M. Armstrong, Henry George, Medical-Officer at Wellington College; Wellington College, Berks. (C. 1900–1, 1901–2.)

1901 Armstrong, John T., Selby House, Beaumont Terrace, West Kensington, W.

O.M. Ashby, Henry, M.D., Physician at The Manchester General Hospital for Children; Lecturer on Diseases of Children at Owens College; 13, St. John Street, Manchester. (W. 1904–5.) (C. 1900–1, 1901–2, 1902–3.)

O.M. Barnardo, Thomas John, 18, Stepney Causeway, E.

1900 Barr, James, M.D., Physician at The Royal Infirmary, Liverpool; 72, Rodney Street, Liverpool.

1902 Barrett, Alfred Keppel, 5, Bedford Gardens, Kensington, W.

1904 Batchelor, Charles, 1, Fairfield Avenue, Staines, Middlesex.

1901 Battams, J. Scott, 123, Lavender Hill, S.W.

1904 Baumann, E. P., M.D., Box 1257, Johannesburg, Transvaal, S. Africa.

O.M. Beach, Fletcher, M.B., Physician at The West-End Hospital for Diseases of the Nervous System; Winchester House, Kingston Hill, Surrey. (C. 1900–1, 1901–2, 1902–3.)

1901 Beddard, Arthur Philip, M.D., Senior Assistant-Physician and Pathologist at The West-London Hospital; 44, Seymour Street, W.

1904 Bidwell, Leonard, Surgeon at The West London Hospital; 15, Upper Wimpole Street, W.

1900 Blagg, Arthur F., M.D., Physician at The Orthopædic Hospital for Children, Bristol; 28, Caledonia Place, Clifton, Bristol.

1902 Blaikie, James Brunton, M.B., Physician at The Hospital for Consumption, Margaret Street, W.; 22, Grosvenor Street, W.

O.M. Bluck, Walter Cardy, M.B., Byford House, York Road, Southend-on-Sea.

O.M. Blumfeld, Joseph, M.D., Assistant-Anæsthetist at St. George's Hospital; 7, Cavendish Place, W. (C.)

Elected

1904 Bolton, Charles, M.D., Assistant-Physician at University College Hospital; Assistant-Physican at The North-Eastern Hospital for Children, Hackney Road, N.E.; 16, Devonshire Street, W.

1903 Braithwaite, John, M.D., Physician at The Devonshire Hospital and Buxton Bath Charity, Hardwick Cliff, Buxton.

O.M. Brightman, Frank, Aspley House, Broadstairs, Kent.

1904 Briscoe, J. Charlton, M.B., Physician at The Evelina Hospital for Sick Children; 110, Harley Street, W.

1901 Brown, John Henry, M.D., 14, Burngreave Road, Sheffield.

O.M. Browne, Hablot J. M., M.B., Hoylake, Cheshire.

1901 Bryant, John Henry, M.D., Assistant-Physician at Guy's Hospital; 8, Mansfield Street, W.

1904 Burgess, Harold, L.

1904 Burgess, Richard.

1901 *Burgess, William Milner, 43, Nicoll Road, Harlesden, N.W. (C. 1904–5.)

O.M. Burghard, Fred. François, M.S., Surgeon at King's College Hospital and at Paddington-Green Children's Hospital; 86, Harley Street, W.

1905 Burnet, James, M.D., Registrar, Royal Hospital for Sick Children, Edinburgh; 20, Polwarth Crescent, Edinburgh.

1904 Burnie, W. Gilchrist, Consulting Medical Officer, Children's Hospital, Bradford; 1, Drewton Street, Bradford.

1904 Burrows, Harold, M.B., Assistant-Surgeon at The Bolingbroke Hospital; 32, Wimpole Street, W.

1901 Burt, Albert Hamilton, 52, Lansdowne Place, Hove, Sussex.

O.M. Burton, Charles George, 77, Church Street, Lower Edmonton, N.

1902 Buzzard, E. Farquhar, M.B., Assistant-Physician at The Belgrave Hospital for Children, Assistant-Physician at The Royal Free Hospital; 33, Harley Street, W.

1902 Bythell, W. J. S., "Woodcliffe," Prestwich, Manchester.

Elected

1905 Campbell, Harry, M.D., Physician at the North-West London Hospital, and at the West End Hospital for Nervous Diseases; 23, Wimpole Street, W.

1902 Campbell, Henry Johnstone, M.D., Senior Physician at The Bradford Royal Infirmary.

1901 Campbell, Robert, M.B., Senior Surgeon at The Belfast Hospital for Sick Children; 21, Great Victoria Street, Belfast. (C. 1904–5.) (C.)

O.M. Carmichael, James, M.D., Consulting-Physician at The Royal Edinburgh Hospital for Sick Children; 22, Northumberland Street, Edinburgh. (C. 1901–2.)

O.M. Carpenter, George, M.D, Physician at The North-Eastern Hospital for Children; late Physician at The Evelina Hospital for Sick Children; Editor and Founder of 'The British Journal of Children's Diseases'; 12, Welbeck Street, W. (C. 1900–1.) (S. 1901–2, 1902–3, 1903–4.) (Ed. 'Reports' 1900–1, 1901–2, 1902–3, 1903–4, 1904–5.) (Ed.)

O.M. Carre-Smith, Herbert L., 74, Holland Park Avenue, Holland Park, W. (C. 1902–3, 1903–4, 1904–5.)

1900 Cathcart, George C., M.B., Senior Assistant-Surgeon at The London Throat Hospital, 35, Harley Street, W.

O.M. *Cautley, Edmund, M.D., Physician at The Belgrave Hospital for Children; Physician at The Metropolitan Hospital; 15, Upper Brook Street, W. (C. 1900–1, 1901–2, 1902–3.) (S. 1904–5.) (S.)

1901 Cave, Edward John, M.D., Physician at The Royal United Hospital, Bath; 20, Circus, Bath.

O.M. Chaffey, Wayland C., M.D., Physician at The Royal Alexandra Hospital for Children, Brighton; 18, Palmeira Square, Hove, Sussex. (C. 1900–1, 1901–2, 1902–3.)

1901 Chaldecott, John Henry, Anæsthetist at St. John's Hospital for Diseases of the Skin, at The Metropolitan Throat, Nose, and Ear Hospital, and at The Italian Hospital; 60, Harley Street, W.

O.M. Chapman, Charles William, M.D., Physician at The Hospital for Diseases of the Heart, Soho Square; 21, Weymouth Street, W. (C. 1903–4, 1904–5.) (C.)

Elected

1901 Cheyne, William Watson, C.B., C.M., F.R.S., Surgeon at King's College Hospital, and at Paddington-Green Children's Hospital; 75, Harley Street, W. (C. 1901–2.)

1901 Clapham, E. W., 48, Harley Street, W.

O.M. Clarke, Ernest, M.D., Surgeon at The Central London Ophthalmic Hospital; Ophthalmic-Surgeon at The Miller Hospital, Greenwich; 3, Chandos Street, Cavendish Square, W.

O.M. Clarke, James Jackson, M.B., Surgeon to Out-patients at The City Orthopædic and at The North-West-London Hospitals; 18, Portland Place, W.

1904 Clogg, H. S., M.S., Assistant-Surgeon at Charing Cross Hospital, and Surgeon at the Evelina Hospital for Sick Children; 117A, Harley Street, W.

1902 Cockburn, Robert P., 64, The Avenue, Ealing, W.

1902 Collier, H. Stansfield, Surgeon to Out-patients at St. Mary's Hospital, and Assistant-Surgeon at The Hospital for Sick Children, Great Ormond Street; 57A, Wimpole Street, W.

1901 Collier, Mark Purcell Mayo, M.S., Senior Assistant-Surgeon at The North-West-London Hospital; Consulting-Surgeon at The National Hospital for Diseases of the Nervous System; 133, Harley Street, W.

1901 Cooper, Percy Robert, M.D., "Glenthorne," The Downs, Bowdon, Cheshire.

1901 Corner, Harry, M.D., Brook House, Southgate, N.

1905 Cowper, W. P., 30, Frognal Lane, Hampstead, N.W.

1901 Cox, Joshua J., M.D., Physician for Diseases of Children at The Clinical Hospital, Manchester; 38, Deansgate, Manchester.

1904 Critchley, Harry George, M.D., 10, Bentinck Street, W.

1902 Cross, Ernest, W., "The Limes," Wallwood Park, Leytonstone.

1901 Crossley, Leonard, M.B., "Beechwood," Arnside, Carnforth, Lancashire.

1902 Crowley, Ralph H., M.D., Physician at The Bradford Royal Infirmary; 116, Manningham Lane, Bradford.

Elected

1904 Curl, Sydney Walter, M.D., 58, Crouch Street, Colchester.

1902 Curtis, Henry J., B.S, late Surgeon at The Royal Hospital for Women and Children; late Surgeon at the North-Eastern Hospital for Children: Surgeon at the Memorial Hospital, Bulawayo, S. Africa.

1901 Dawnay, Archibald Hugh Payan, Anæsthetist at The Royal Orthopædic Hospital; 67a, Harley Street, W.

O.M. Deas, Frank, Thorndale, Merton Park, S.W. (C.)

O.M. Dent, Clinton T., M.C., Surgeon at St. George's Hospital and at The Belgrave Hospital for Children; 61, Brook Street, W. (C. 1902–3, 1903–4, 1904–5.)

O.M. Dockrell, Morgan, M.D., Physician at St. John's Hospital for Diseases of the Skin; 9, Cavendish Square, W.

1901 Dodd, Arthur Herbert, 49, Church Road, Hove, Sussex.

1901 Douglas, Arch. R., Assistant-Medical Officer and Deputy Superintendent at The Royal Albert Asylum, Lancaster.

1901 Drake-Brockman, E. F., Lieutenant-Colonel I.M.S., late Professor of Physiology and Diseases of the Eye at The Medical College, Madras, and late Superintendent at The Eye Infirmary, Madras, India; 14, Welbeck Street, W.

O.M. Drew, Douglas, B.S., Surgeon at The North-Eastern Hospital for Children and at The Hospital for Women, Soho Square; 1, Harley Street, W.

1905 Dudgeon, Leonard S., Superintendent of Clinical Laboratory, St. Thomas's Hospital; 6, Powis Gardens, Bayswater, W.

1901 Dun, Robert Craig, M.B., Surgeon at The Liverpool Infirmary for Children; 6, Rodney Street, Liverpool.

1901 Dunlop, James, Aveley, Purfleet, Essex.

1901 Dunn, W. J. Robertson, 46, Wellington Terrace, West Derby Road, Liverpool.

1901 Edelsten, Ernest Alfred, M.B., 370, Brixton Road, S.W.

1904 Edmunds, Arthur, B.S., Assistant-Surgeon at The Paddington Green Children's Hospital; 23, Upper Wimpole Street, W.

Elected

O.M. Edmunds, Walter, M.C., Surgeon at The Tottenham Hospital; 2, Devonshire Place, W. (C. 1900–1, 1901–2, 1902–3.)

1905 Elliott, Andrew, M.D., Medical Registrar, West London Hospital; 11, Upper Berkeley Street, Portman Square, W.

1901 Ellis, W. McDonogh, M.D., Assistant-Physician at The Hospital for Women, Bath; Cedar Lodge, Bath.

1904 Emanuel, J. G., M.D., Physician to Out-patients at Queen's Hospital, Birmingham; 47, Newhall Street, Birmingham.

1905 English, T. Crisp, B.S., Assistant-Surgeon at St. George's Hospital, and at the Grosvenor Hospital for Women and Children; 31, Grosvenor Street, W.

1904 Evans, Arthur, M.S., Assistant-Surgeon at Westminster Hospital, Surgeon to out-patients at The Seamen's Hospital, Royal Victoria and Albert Dock; 53, Queen Anne Street, W.

1901 Evans, J. Howell, M.Ch., 31, New Cavendish Street, Portland Place, W.

1901 Evans, Thomas Hy., 95, Shaw Street, Liverpool.

1904 Evelyn, William Arthur, M.D., 24, Micklegate, York.

O.M. Ewart, William, M.D., Senior Physician at St. George's Hospital and at The Belgrave Hospital for Children; 33, Curzon Street, Mayfair, W. (C. 1904–5.) (C.)

O.M. Fenwick, William Soltau, M.D., Senior Physician at The London Temperance Hospital, late Physician at The Evelina Hospital for Sick Children; 29, Harley Street, W.

1901 Fisher, H. Wycliffe, "Rathescar," Greenbank Drive, Sefton Park, Liverpool.

O.M. Fisher, Theodore, M.D., late Pathologist at The Bristol Royal Infirmary, and late Physician to Out-patients at The Bristol Hospital for Sick Children; Pathologist at The North-Eastern Hospital for Children. (C. 1900–1.) (P.S. 1901–2, 1902–3, 1903–4.) (C. 1904–5.)

1902 Flower, George John William, "Hendford," Yeovil, Somerset.

Elected

1901 Forster, Henry John, 119, Victoria Road, Romford, Essex.

1903 Fortescue-Brickdale, John Matthew, M.D., Registrar and Pathologist at The Royal Hospital for Children and Women, Bristol; 52, Pembroke Road, Clifton, Bristol.

O.M. Fox, T. Colcott, M.B., Physician for Diseases of the Skin at Westminster Hospital, and at Paddington-Green Hospital for Children; 14, Harley Street, W.

1901 Freeland, Ernest Harding, Surgeon at St. George's and St. James's General Dispensary; 145, Harley Street, W.

1901 *Frost, Edmund, M.B., "Chesterfield," Chesterfield Road, Eastbourne.

O.M. Fuller, Andrew, Local Government Board Inspector; "The Bushes," Chase Road, Oatlands Park, Weybridge, Surrey.

1901 George, Alfred Walter, M.D., Surrey House, Shootup Hill, N.W.

1905 Gerrard, Alfred H., M.B., Surgeon at the South Wimbledon and Merton Cottage Hospital; 90, Merton Road, Wimbledon, S.W.

1904 Gerrish, Daniel S., Coningsby Villa, 332, Church Road, St. George, Bristol.

1900 Gervis, F. Harcourt, M.D., 1, Lyncroft Mansions, West Hampstead, N.W.

1905 Gibson, George A., M.D., Physician at the Royal Infirmary, Edinburgh; 3, Drumsheugh Gardens, Edinburgh. (C.)

O.M. Giffard, Henry Edward, Denham House, Egham.

O.M. Giles, Leonard Thomason, 5, St. Nicholas Parade, Scarborough.

1905 Goldsmith, G. Harvey, M.D., Ophthalmic Surgeon, at Bedford County Hospital; Harpur Place, Bedford.

1901 Grant, J. Dundas, C.M., Surgeon at The Central London Throat and Ear Hospital; 18, Cavendish Square, W.

O.M. Gray, Alexander C. E., M.D., 1, Elm Park Gardens, South Kensington, W.

Elected

1904 Grünbaum, Otto F. F., M.D., Assistant-Physician at the Belgrave Hospital for Children; Medical Registrar at the London Hospital; 30a, Wimpole Street, W.

1901 Gullan, Archibald Gordon, M.D., 5, Canning Street, Liverpool.

O.M. Guthrie, Leonard George, M.D., Physician at The Hospital for Epilepsy and Paralysis, Regent's Park; Senior Physician at The Paddington-Green Children's Hospital; 15, Upper Berkeley Street, W. (C. 1900–1, 1901–2, 1902–3.) (C. 1904–5.) (C.)

O.M. Gwynne, Charles Nelson, M.Ch., Surgeon at The Children's Hospital, Sheffield; Highfield House, Sheffield. (C. 1903–4, 1904–5.) (C.)

1904 Harke, S. L., M.B.

1904 Harman, N. Bishop, M.B., Ophthalmic Surgeon, Belgrave Hospital for Children; 108, Harley Street, W.

O.M. Hawken, Giles Lockwood Lang, Surgeon at St. John's College, Hurstpierpoint, Sussex.

1901 Hawthorne, Charles Oliver, M.D., Physician at The Central London Ophthalmic Hospital; Assistant-Physician at The Royal Hospital for Children and Women; 63, Harley Street, W. (C.)

1901 Hayes, Thomas C., M.D., Professor of Obstetric-Medicine and Diseases of Women at King's College Hospital; 17, Clarges Street, Mayfair, W.

1904 Haynes, G. Secretan, 4, Trinity Street, Cambridge.

1901 Heywood, Charles Christopher, M.B., Winter's Buildings, St. Ann Street, Manchester.

1904 Hill, G. Wm., M.D., Surgeon for Diseases of the Ear at St. Mary's Hospital; 26, Weymouth Street, Portland Place, W.

1902 Hinds, Frank, M.D., Honorary-Surgeon at The Infirmary, Worthing; 17, Tennyson Road, Worthing. (C. 1904–5.) (C.)

1901 Hobhouse, Edmund, M.D., Assistant-Physician at The Sussex County Hospital; Physician at The Royal Alexandra Hospital for Children, Brighton; 12, Second Avenue, Hove. (C. 1903–4.) (C.)

Elected

1904 Hobson, H. Overton, M.D., 32, Upper Berkeley Street, W.

1905 Hollings, Guy B., M.D., 22, Endsleigh Gardens, N.W.

1901 Hopkinson, Albert, M.B., 2, Parsonage Road, Withington, Manchester.

1904 Howard, John Alexander, M.D., 40, Harold Road, Upper Norwood.

1901 Hutchison, Robert, M.D., Assistant-Physician at The London Hospital, and at The Hospital for Sick Children, Great Ormond Street; 22, Queen Anne Street, W. (C. 1901–2, 1902–3, 1903–4.)

O.M. Hutton, David Mount, M.D., 5, Lulworth Road, Birkdale.

O.M. Hutton, Henry Richmond, M.B., Physician at The Manchester General Hospital for Children; 16, St. John Street, Manchester. (C. 1903-4, 1904–5.) (C.)

1901 Huxley, Henry, 39, Leinster Gardens, Hyde Park, W.

1904 imThurm, Robert McKenzie, Ailsa, Sydenham.

O.M. Jaffrey, Francis, Assistant-Surgeon at St. George's Hospital; Surgeon to Out-patients at The Belgrave Hospital for Children; 33, Nottingham Place, W. (C. 1901–2, 1902–3, 1903–4.) (S.)

1901 James, Charles Alfred, 49, Stamford Hill, N.

O.M. Jessop, Walter Hamilton Hylton, M.B., Ophthalmic-Surgeon at St. Bartholomew's Hospital; 73, Harley Street, W.

1901 Johnson, L. Capper, Lidgate Park Road, Roundhay, Leeds.

1902 Johnson, Raymond, Surgeon at University College Hospital and at The Victoria Hospital for Children; 11, Wimpole Street, W.

1904 Jones, A. Ernest, M.D., 13, Harley Street, W.

O.M. Jones, Robert, Surgeon at The Royal Southern Hospital, Liverpool; 11, Nelson Street, Liverpool. (C. 1900–1, 1901–2.)

1903 Jones, W. Llewellyn, M.D., 58, Thomas Street, Merthyr Tydfil, South Wales.

1901 Kapadia, S. A., M.D., 49, Longridge Road, South Kensington.

Elected

1905 Kellie, Kenneth H. A., M.B., Westfield, Curzon Park, Chester.

1902 Kelson, William Hy., M.D., Assistant-Surgeon at The London Throat Hospital; 17, Cavendish Place, Cavendish Square, W.

O.M. Knapton, George, Yew Bank, Bowdon, Cheshire.

1904 Knight, Frederick, M.D., Physician at The Swansea Hospital; 10, Walter Terrace, Swansea.

O.M. Lack, Harry Lambert, M.D., Surgeon to the Throat Department at The London Hospital; Surgeon at The Throat Hospital, Golden Square; 48, Harley Street, W.

1901 Lacy, Alexander Gairdner, "Saumarez," Sunninghill, Ascot, Berks.

1901 Lake, Richard, Surgeon at The Royal Ear Hospital; 60, Harley Street, W.

O.M. Lambert, Sydney H. A., M.B., Gothic House, Harrow-on-the-Hill.

1901 Langdon-Down, Percival L., M.B., 47, Welbeck Street, W.

1900 Langdon-Down, Reginald Langdon, M.B., 47, Welbeck Street, W.

O.M. Lankester, Alfred Owen, 5, Upper Wimpole Street, W.

O.M. Lawson, Arnold, M.D., Assistant-Surgeon at The Royal London Ophthalmic Hospital, and Ophthalmic-Surgeon at The Children's Hospital, Paddington-Green; 12, Harley Street, W.

O.M. Lee, Philip George, Surgeon at The Women and Children's Hospital, Cork; 25, St. Patrick's Hill, Cork.

1904 Leedham-Green, Charles A., M.D., Surgeon at Queen's Hospital, Birmingham; 9, Newhall Street, Birmingham.

1901 Leslie, W. Murray, M.D., 74, Cadogan Place, Belgrave Square, S.W.

1904 Lett, Hugh, Ch.B., Assistant-Surgeon at The Belgrave Hospital; 25, Queen Anne Street, W.

O.M. Lewis, Percy G., M.D., Honorary Medical Officer at The Victoria Hospital, Folkestone; 22, Manor Road, Folkestone. (C. 1903–4, 1904–5.) (C.)

Elected

1904 Lingard, Alfred, M.B., Imperial Bacteriologist to the Indian Government, Imperial Bacteriological Laboratory, Nainital District, United Provinces, India.

O.M. Lister, Thomas David, M.D., Assistant-Physician at the Royal Hospital for Women and Children, Assistant-Physician at The Mount Vernon Hospital; 50, Brook Street, W.

1904 Lister, William Tindall, B.C., Ophthalmic-Surgeon at The Hospital for Children, Great Ormond Street, Assistant-Surgeon at The Central London Ophthalmic Hospital; 30, Queen Anne Street, W.

1901 Livingstone, George R., 35, George Street, Dumfries, N.B.

1905 Low, V. Warren, B.S., Assistant-Surgeon at St. Mary's Hospital; 27, Queen Anne Street, Cavendish Square, W.

O.M. *Lucas, R. Clement, B.S., Senior Surgeon and Lecturer on Surgery at Guy's Hospital; Consulting-Surgeon at The Evelina Hospital for Sick Children; 50, Wimpole Street, W. (T.) (W. 1903–4.) (C.C.)

1905 Maddox, R. H., M.B., Major I.M.S., Ranchi, Chota Nagpur, India.

O.M. McCaw, John, M.D., Physician at The Belfast Hospital for Children; 78, Dublin Road, Belfast. (C. 1901–2.)

1904 McGregor, John, M.D., 15a, Pembridge Crescent, W.

1905 McIlraith, C. H., M.D., 44, Half Moon Street, Herne Hill, S.E.

O.M. McKenzie, Dan, M.D., Holly Lodge, Leytonstone.

O.M. McKerron, Robert Gordon, M.D., Senior Physician at The Children's Hospital, Aberdeen; 1, Albyn Place, Aberdeen. (C. 1902–3.)

1901 McNab, Alexander Anderson, M.B., 15, Wesley Road, Armley, Leeds.

1900 Macalister, Charles John, M.D., Physician at The Liverpool Royal Southern Hospital; 35, Rodney Street, Liverpool. (C. 1901–2, 1902–3, 1903–4.)

1901 Mackintosh, John Stewart, St. Ive, Platt's Lane, Hampstead, N.W.

1901 Mackintosh, William Alexander, M.B., 3, Park Terrace, Stirling. (C. 1904–5.)

Elected

1901 Maitland, Alfred Derwent, 1, Gloucester Villas, Hanwell, W.

1904 Makin, Frank Humphrey, M.B., c/o 'South African Register,' 80, Fleet Street, E.C.

1901 Makins, George Henry, C.B., Surgeon at St. Thomas's Hospital; Consulting-Surgeon at The Evelina Hospital for Sick Children; 47, Charles Street, Berkeley Square, W.

O.M. Malcolm, John D., C.M., Surgeon at The Samaritan Free Hospital; 13, Portman Street, Portman Square, W.

1904 Marsh, J. J., 104, Sloane Street, S.W.

O.M. Marshall, Lewis W., M.D., Surgeon at The Children's Hospital, Nottingham; 42, The Ropewalk, Nottingham. (C. 1900–1, 1901–2, 1902–3.)

1900 May, W. Page, M.D., 9, Manchester Square, W.

1920 Maynard, J. Seymour, M.B., 4, Charles Street, St. James's Square, S.W.

1905 Mayou, Marmaduke S., 46, Weymouth Street, Portland Place, W.

1904 Midelton, William John, 72, Charminster Road, Bournemouth.

O.M. Milburn, Charles Henry, M.S., Surgeon at The Victoria Hospital for Children, Hull; 158, Anlaby Road, Hull.

1902 Miller, Charles Hewitt, Oughtrington House, Lymm, Cheshire.

1902 Milligan, Wyndham Anstruther, C.M., Anæsthetist at The Great Northern Central Hospital; 104, Bethune Road, N.

1901 Milne, Robert, M.D., 32, Bow Road, E.

O.M. Moline, Paul F., M.B., Anæsthetist at The Victoria Hospital for Children, Chelsea; 42, Walton Street, Chelsea, S.W.

1901 Monsarrat, Keith Waldegrave, C.M., Surgeon at The Northern Hospital, Liverpool; 11, Rodney Street, Liverpool. (C. 1904–5.)

1901 Montgomery, W. P., B.S., Surgeon at The Children's Hospital, Manchester, Assistant-Surgeon at The Manchester Royal Infirmary; 15, St. John Street, Manchester.

Elected

1901 Morgan, Cecil Augustus, M.B., 6, Somerset Street, Portman Square, W.

1902 Morgan, George, Senior Surgeon at The Children's Hospital, Brighton; 6, Pavilion Parade, Brighton.

1901 Morison, Alexander, M.D., Physician to Out-patients at The Great Northern Central Hospital, and at The Children's Hospital, Paddington-Green; 14, Upper Berkeley Street, W. (C. 1903–4, 1904–5.) (C.)

1904 Morton, C. A., Surgeon at The Bristol General Hospital, and at The Bristol Royal Hospital for Sick Children; 14, Vyvyan Terrace, Clifton.

O.M. Mossop, Isaac, Surgeon at The Bradford Children's Hospital; Victor Lodge, Manningham, Bradford.

1905 Mounsey, R. E., Moorlands, Camberley, Surrey.

1902 Mummery, John Percy Lockhart, B.C., 10, Cavendish Place, W., Assistant-Surgeon at The North-Eastern Hospital for Children. (C. 1904–5.) (C.)

1901 Munro, Henry Acland, M.B., "Lulworth," Rushey Green, Catford, S.E.

1904 Murphy, J. Keogh, B.C., Surgeon to Out-patients at The Paddington Green Children's Hospital; 9, Weymouth Street, Portland Place, W.

1901 Murtz, Anton Peter, Clareville, 48, Queen's Road, Finsbury Park, N.

1901 Nabarro, David N., M.D., Assistant-Professor of Pathology and Lecturer on Bacteriology, University College; 6, Crown Office Row, Inner Temple, E.C.

1904 Nash, Elwin H. T., Oulton House, Burnley Road, Accrington, Lancs.

1901 Nash, W. Gifford, Surgeon at The Bedford County Hospital; Clavering House, Bedford.

1902 Newbolt, George P., Surgeon at The Royal Southern Hospital, Liverpool; 42, Catherine Street, Liverpool.

1901 Nisbet, J. Tawse, M.D., 7, Croxteth Road, Liverpool.

1901 Niven, Robert, Camberwell Infirmary, Brunswick Square, S.E.

1902 Northcote, Percy, M.B., 101, Jermyn Street, S.W.

Elected

1901 Oldmeadow, Lloyd J. H., M.D., Kineton, Warwickshire.

1901 Oppenheimer, Heinrich, M.D., 63, Finsbury Pavement, E.C.

1904 Orme, Gilbert Edward, B.C., Lowfield, Clitheroe, Lancs.

1904 Page, H. M., 14, Greville Place, S.W.

1901 Palmer, Frederick S., M.D., 7, Gordon Place, Gordon Square, W.C.

O.M. Parkinson, J. Porter, M.D., Physician at The London Temperance Hospital and Physician at The North-Eastern Hospital for Children; 57, Wimpole Street, W. (C. 1903–4, 1904–5.) (C.)

1902 Parkinson, Thomas W., M.D., 77, Sloane Street, S.W.

1903 Parry, Robert H., Surgeon at The Victoria Infirmary and at The Royal Hospital for Sick Children, Glasgow; 3, Royal Crescent, Queen's Park, Glasgow. (C.) (1903–4.)

1901 Paterson, Alexander Gordon, M.D., South Lodge, Ascot, Berks.

1903 Pearson, S. Vere, M.B., Assistant-Physician at The East London Hospital for Children, Shadwell; 8, Serjeant's Inn, E.C.

1901 Pedley, R. Denison, Dental-Surgeon at The Evelina Hospital for Sick Children, S.E.; 17, Railway Approach, London Bridge, S.E.

1901 Pendlebury, Herbert S., B.C., Surgeon at The Royal Hospital for Women and Children, Assistant-Surgeon at St. George's Hospital; 44, Brook Street, W.

O.M. *Penrose, Frank, M.D., Physician at St. George's Hospital and at The Hospital for Children, Great Ormond Street; Penmener, Parkstone, Dorset.

1901 Pernet, George, Assistant at the Skin Department, University College Hospital; 94, Harley Street, W. (C. 1903–4, 1904–5.) (C.)

1901 Perry, Herbert, M.D., Assistant-Physician at The General Hospital, Birmingham, Physician to Outpatients at The Children's Hospital, Birmingham; 54, Newhall Street, Birmingham. (C.)

O.M. Peskett, A. W. Chalmers, M.B., 4, Clermont Road, Preston Park, Brighton.

Elected

1902 Phillips, Hugh, M.B., 28, Palace Court, W.

1905 Pike, Douglas R., M.B., 3, Cromwell Crescent, Kensington, S.W.

1904 Piper, Francis Perris, M.B., King's Leigh, Tankerton, Whitstable.

1901 Poland, John, Senior Surgeon at The Miller Hospital, Greenwich; Surgeon at The City Orthopædic Hospital; 2, Mansfield Street, W.

1902 Potter, Bernard Elwell, M.B., Resident Medical Officer at St. George's Dispensary, Hanover Square: 16, Little Grosvenor Street, W.

1901 Potts, William A., M.D., 160, Hagley Road, Birmingham.

1904 Powell, Hugh Falkenberg, 1, Seagrove Place, Pittville, Cheltenham.

1904 Poynton, Frederick J., M.D., Assistant-Physician at University College Hospital, and at the Hospital for Sick Children, Great Ormond Street; 1, Harley Place, Harley Street, W.

O.M. *Prangley, Henry J., Tudor House, Anerley, S.E.

1901 Pratt, Eldon, M.D., Henfield, Sussex.

1901 Pritchard, George Eric Campbell, M.D., 14, Cromwell Place, South Kensington, S.W.

1901 Quennell, Arthur, Brentwood, Essex.

1905 Randall, Martin, M.D., The Lodge, Leopold Road, Wimbledon, S.W.

1901 Raw, Nathan, M.D., Mill Road, Liverpool.

1901 Ray, John Howson, Ch.M., Assistant-Surgeon at The Salford Royal Hospital; Surgeon at The Children's Hospital, Pendlebury; 22, St. John Street, Manchester.

1901 Reeves, Henry A., Surgeon at The Hospital for Women and at The Royal Orthopædic Hospital; 70A, Grosvenor Street, W.

1901 Rendel, Arthur Bowen, M.B., 43, Albion Street, Hyde Park, W.

1901 Rhodes, John Milson, M.D., Didsbury.

1905 Richardson, William G., B.S., Assistant-Surgeon at Newcastle-on-Tyne Royal Infirmary; 19, Saville Row, Newcastle-on-Tyne.

Elected

1903 Ridley, J. Brooke, M.D., 32, Sydenham Road, Croydon.

1901 Roberts, George A., Oaklands, Langton, Tunbridge Wells.

O.M. Robinson, Henry Betham, M.S., Assistant-Surgeon at St. Thomas's Hospital and Surgeon at The East-London Hospital for Children; 1, Upper Wimpole Street, W. (C. 1901–2, 1902–3, 1903–4.)

O.M. Rogers, Bertram, M.H., M.D., Physician at The Hospital for Sick Children and Women, Bristol; 1, Victoria Square, Clifton, Bristol.

1901 Rose, Samuel Frank, Assistant-Surgeon at The National Dental Hospital; Dental-Surgeon at The North-Eastern Hospital for Children; 59, Queen Anne Street, W.

O.M. Roth, Bernard, Orthopædic-Surgeon at The Royal Alexandra Hospital for Children, Brighton; 38, Harley Street, W.

1902 Rowlands, Robert Pugh, B.S., The College, Guy's Hospital, Assistant Surgeon at The East London Hospital for Children.

1902 *Roxburgh, Alex. Bruce, B.Ch., Surgeon at The Poplar Hospital, and Assistant-Surgeon at The London Hospital; 7, Henrietta Street, Cavendish Square, W.

O.M. Rugg, George L., 106, Knight's Hill Road, West Norwood, S.E.

1901 Rumboll, Sydney, Cavendish House, Leeds.

1904 Sale-Barker, Horace John Maurice Drummond, Medical Officer in Charge X-ray and Electrical Departments at The North-Eastern Hospital for Children, and at Westminster Hospital; 45, Hill Street, Knightsbridge.

O.M. *Sansom, A. Ernest, M.D., Consulting-Physician at The London Hospital, and Consulting-Physician at The North-Eastern Hospital for Children; "Beversbrook," Finchley Road, Hampstead, N.W. (C.C. 1900–1, 1901–2.) (C. 1902–3.)

O.M. Sansom, Henry Arthur, M.D., 127, West End Lane N.W.

1904 Sawyer, James E., M.D., 93, Cornwall Street, Birmingham.

Elected

1901 Schilling, George.

1904 Scofield, H. C. Lingham, M.B., "Northwood," Dulwich Grove, North Dulwich, S.E.

1904 Scott, W. J., M.B., Clifton House, Sheet Street, Windsor.

O.M. Sequeira, James H., M.D., Physician in Charge of the Skin Department at The London Hospital; 63, Harley Street, W.

1901 Shackleton, William Webb, M.B., Grove House, Bushey, Herts.

O.M. Shuttleworth, George E., M.D., late Medical Superintendent Royal Asylum for Imbeciles, Lancaster; Medical Examiner defective children, Willesden School Board; Medical Expert (M.A.B.) Rochester House Inst., Ealing; Ancaster House, Richmond Hill, Surrey. (C. 1900–1, 1901–2, 1902–3.)

1901 Simson, Henry J. Forbes, 80, Brook Street, W.

1901 Skelding, Henry, B.C., Surgeon at The Bedford County Hospital; St. Loyes, Bedford. (C. 1904–5.) (C.)

1901 *Smith, Frederick John, M.D., Physician at The London Hospital; 138, Harley Street, W.

1901 Smith, Guy Bellingham, B.S., Assistant-Obstetric Physician, Guy's Hospital; 24, St. Thomas's Street, S.E.

1901 *Smith, Travers, R.M., M.B., 13, Kilburn Park Road, N.W.

1904 Smith, William R., M.D., Professor of Forensic Medicine, King's College, London; 74, Great Russell Street, Bloomsbury Square, W.C.

1901 Smyth, Sidney B., M.B., Physician at The Hospital for Sick Children, Belfast; Ulsterville House, Lisburn Road, Belfast. (C. 1903–4.)

O.M. Snape, Ernest, M.D., 41, Welbeck Street, W.

O.M. Spokes, P. Sidney, Dental-Surgeon at University College Hospital; 4, Portland Place, W.

1903 Spriggs, Edmund Ivens, M.D., Physician to Outpatients at The Victoria Hospital for Children and at The City of London Chest Hospital; 24, St. Thomas's Street, S.E.

Elected

1904 Spurway, John, M.B., 29, Brondesbury Road, Kilburn, N.W.

O.M. Staunton, Michael Cornelius, M.D., Visiting-Surgeon at The Children's Hospital, Dublin; 22, North Frederick Street, Dublin.

1901 Staveley, William H. C., 24, Sloane Gardens, S.W.

O.M. Stephenson, Sydney, C.M., Ophthalmic-Surgeon at The Evelina and The North-Eastern Hospitals for Children; Editor and Founder of 'The Ophthalmoscope'; 33, Welbeck Street, W. (S. 1900–1, 1901–2, 1902–3.) (C. C. 1903–4, 1904–5.)

O.M. Stewart, Hugh M., M.D., Walton House, Lordship Lane, S.E.

O.M. Stiles, Harold J., C.M., Surgeon at The Royal Edinburgh Hospital for Sick Children; 8, St. Colme Street, Edinburgh. (C. 1900–1.) (C. 1902–3, 1903–4.)

1900 Street, Alfred Francis, M.D., "Burghfield," St. Mildred's Road, Westgate-on-Sea.

O.M. Summerhill, Thomas Henry, Surgeon at The Alexandra Children's Hospital, Rhyl; "Bodfor," Rhyl, Flintshire.

O.M. Sunderland, Septimus P., M.D., Physician at The Royal Hospital for Children and Women; Obstetric-Physician at the French Hospital; 11, Cavendish Place, W.

O.M. Sutherland, George A., M.D., Physician at The Paddington-Green Children's Hospital; Assistant-Physician at The North-West-London Hospital; 73, Wimpole Street, W. (C. 1900–1, 1901–2, 1902–3.) (S. 1903–4, 1904–5.) (C.)

1904 Swainson, James Midleton Graham, Assistant-Surgeon North-Eastern Hospital for Children.

1901 Swan, Russell H. J., M.B., 8, St. Thomas's Street, London Bridge, S.E.

1901 Swanton, James Hutchinson, M.D., 40, Harley Street, W.

1901 *Targett, James H., M.S., Assistant-Obstetric-Surgeon at Guy's Hospital; 19, Upper Wimpole Street, W.

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1901 *Taylor, Frederick, M.D., Senior Physician at Guy's Hospital; Consulting-Physician at The Evelina Hospital for Sick Children; 20, Wimpole Street, W. (C. 1901–2.) (C. 1903–4.)

O.M. Taylor, James, M.D., Physician at The National Hospital for Paralysed and Epileptic, Queen Square, and Physician at The North-Eastern Hospital for Children; 49, Welbeck Street, W. (C. 1901–2, 1902–3, 1903–4.)

1904 Thomas, William, B.S., Surgeon at the Orthopædic Hospital, Birmingham; 200, Bristol Road, Birmingham.

1902 Thorne, Atwood, M.B., Assistant-Surgeon at The London Throat Hospital; 148, Harley Street, W.

1901 Thorne, W. Bezly, M.D., 53, Upper Brook Street, W.

1902 Tod, Hunter, B.C., 101, Harley Street, W., Aural-Surgeon at The London Hospital.

1901 Trout, J. H. Ensor, 412, Monument Road, Birmingham.

O.M. Tubby, Alfred H., M.S., Surgeon at Westminster Hospital; Consulting-Surgeon at The Evelina Hospital for Sick Children; Surgeon at The National Orthopædic Hospital; 25, Weymouth Street, W. (C. 1900–1, 1901–2.) (C.C. 1902–3.) (C. 1904–5.) (C.)

1903 Tullis, George Augustus, M.D., Holmbush, Grove Road, Southsea.

1901 Turnbull, George Lindsay, M.D., 76, Ladbroke Square, W.

1901 Turner, H. Stanley, "Balgownie," 211, Bridge Road, Battersea, S.W.

1904 Turner, Philip, M.S., Assistant-Surgeon at The Belgrave Hospital for Children; 8, St. Thomas's Street, S.W.

O.M. Vacher, Francis. Consulting-Surgeon at The Wirrell Hospital for Children; "Glenmore," Shrewsbury Road, Birkenhead.

O.M. Vickery, William H., Surgeon at The Children's Hospital, Newcastle-on-Tyne; 52, Elswick Road, Newcastle-on-Tyne. (C. 1900–1.)

1902 Walker, J. W. Thomson, C.M., Assistant-Surgeon at The North-West London Hospital, Assistant-Surgeon at St. Peter's Hospital for Stone; 8, Cavendish Place, W. (C. 1903–4, 1904–5.) (C.)

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1901 Wallace, Joseph B., M.B., 39, Battersea Rise, S.W.

1901 Walsh, David, M.D., Physician at The Western Skin Hospital; Editor of 'The Medical Press and Circular'; 70A, Grosvenor Street, W.

1901 Waring, John Alfred, M.B., 21, Derby Road, Nottingham.

1905 Webb, F. C. Apthorpe, Grafton House, Cambridge.

1901 *Weber, F. Parkes, M.D., Physician at The German Hospital, Dalston; Assistant-Physician at The North-London Hospital for Consumption; 19, Harley Street, W. (C.)

1902 Westmacott, Frederick H., Aural-Surgeon at the Manchester Children's Hospital, Pendlebury; 8, St. John Street, Manchester.

1901 Whipham, Thomas Rowland C., M.B., Assistant-Curator of the Museum at St. George's Hospital, Physician to Out-patients at the Evelina Hospital for Children; 37, Park Street, W.

1901 Whitelocke, Richard H. A., C.M., Surgeon at The Radcliffe Infirmary, Oxford; 6, Banbury Road, Oxford. (C. 1901–2, 1902–3, 1903–4.) (P.S. 1904–5.) (P. S.)

O.M. Wilks, Joseph Henry, B.C., Surgeon at The Children's Hospital, Sheffield; "Fairfield," Sharrow, Sheffield.

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O.M. *Williams, Dawson, M.D., Physician at The East-London Hospital for Children, Editor of 'The British Medical Journal'; 2, Agar Street, Strand, W.C. (C. 1900–1, 1901–2.)

O.M. Williams, Edward Cecil, M.B., Physician to Out-patients at The Bristol Royal Hospital for Sick Children; 9, Whatly Road, Clifton, Bristol.

O.M. Willey, Charles H., C.M., Surgeon at The Sheffield Children's Hospital; Wostenholm Road, Sharrow, Sheffield. (P.S. 1900–1.) (C. 1901–2.)

1904 Willis, Charles Faircourt, Lieut.-Colonel, I.M.S., M.D., "Felixstowe," The Avenue, Upper Norwood, S.E.

1904 Wilson, Geoffrey R., Ongar, Essex.

O.M. Wilson, Kemlo R. M., M.D., Hanwell, W.

Elected

1905 Wilson, R. Mervyn, M.D., Surgeon, Derbyshire Hospital for Sick Children; Full Street, Derby.

1902 Wood, Edward, Stamford Lodge, Waverley Road, Enfield, Middlesex.

1901 Woods, John Francis, M.D., Medical-Superintendent at The Hoxton House Asylum, N.; 29, Queen Anne Street, W.

1901 Wright, Douglas, M.D., 229, Camden Road, N.

1902 Wright, George A., M.B., Surgeon at The Manchester Royal Infirmary; Surgeon at The Children's Hospital, Pendlebury, Manchester; 16, St. John Street, Manchester.

1904 Wylie, Andrew, M.D., 7, Harley Street, W.

*** *It is requested that any inaccuracy in the foregoing list may be notified to the Honorary Editor of the Reports and also to the Senior London Honorary Secretary.*

LIST OF PLATES.

PLATE		FACE PAGE
I, II.	Illustrating Dr. George Carpenter's case of congenital deformity of the hands	240
III, IV.	Illustrating Dr. George Carpenter's and Mr. S. Stephenson's case of tubercle of the choroid, presenting certain clinical and pathological peculiarities	276

LIST OF FIGURES IN THE TEXT.

FIG. PAGE

1. Illustrating Dr. Otto Grünbaum's case of congenital trophic œdema 7

2. Illustrating Mr. G. Pernet's case of trichotillomania . 29

3, 4. Illustrating Dr. George Carpenter's case of herpes zoster extending to the finger-tips . . 30

5—7. Illustrating Mr. Clement Lucas's case of an ill-developed upper lateral incisor tooth . . 34, 36, 37

8—9. Illustrating Mr. S. Gilford's cases of congenital diaphragmatic hernia 39, 42

10, 11. Illustrating Dr. Harry Campbell's case of progressive palsy of cerebral origin . . 68, 69

12. Illustrating Mr. R. C. Dun's case of congenital deformity of both forearms 198

13. Illustrating Dr. Edmund Cautley's case of verrucose endocarditis of the aortic valves 216

14, 15. Illustrating Dr. George Carpenter's and Mr. P. L. Mummery's case of congenital hypertrophic stenosis of the pylorus . . . 243, 244

16. Illustrating Mr. R. C. Dun's case of median harelip . 295

17—19. Illustrating Mr. C. N. Gwynne's cases of multiple xanthoma tuberosum 319, 320, 321

REPORTS

OF

THE SOCIETY

FOR THE

STUDY OF DISEASE IN CHILDREN.

SESSION of 1904–5.

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1. HYPERTROPHY AFFECTING THE RIGHT LOWER LIMB.

By C. O. HAWTHORNE, M.D.

The boy, aged 12 years, was shown to the Society on February the 21st, 1902 (see 'Reports,' vol. ii, p. 114.)

The patches, previously described as hæmorrhagic, on the front of the leg ulcerated some twelve months ago and have never completely healed. Dr. Hawthorne asked the Society whether, in view of the inconvenience which the lad suffered from the condition of the limb, it was justifiable to advise him to submit to amputation.

(*Exhibited October the 21st*, 1904.)

Discussion.

Mr. Arthur Edmunds thought that the condition was a nævoid one, in which the hyperplasia was probably secondary to some increase in the vascularity of the part. He was of opinion that the papillary growths around the ulcer were due to inflam-

matory blocking of the lymphatics in that region. Although he believed that for ulcer of the leg amputation was too rarely performed, in the present case the boy was too young to realise what amputation meant, and the loss of a limb always greatly militated against obtaining employment. However efficient a man might be, the loss of a limb often led to his rejection. The boy had apparently been lying in bed, but probably under very unfavourable conditions, and it was doubtful whether the strict rest enjoined was ever actually obtained. If the patient could be in hospital or under close supervision, with his leg elevated, and at rest with suitable topical treatment, the ulcer would probably be found to heal, and he advocated a prolonged trial of hospital treatment before amputation.

2. A CASE OF HABIT SPASM.

By ROBERT HUTCHISON, M.D.

The spasm in this patient, a boy, aged 10 years, affected chiefly the muscles of the right side of the neck, though there were occasional twitchings of the eyes and violent bowing movements as well. The spasm had been present for two years and had resisted all treatment. There was no history of chorea and no cause was assigned for the onset. Another boy at his school was stated to have developed similar movements as the result of imitation.

(*Exhibited October the* 21*st*, 1904.)

Discussion.

Mr. Donald Armour had seen cases of habit spasm in Queen's Square Hospital which were treated by hypnotism, but without the slightest result.

Dr. G. A. Sutherland said he had read in one of Dr. Cheadle's writings that in cases of habit spasm of that kind it passed off on the completion of the second dentition. Dr. Cheadle did not recommend any treatment, and his (Dr. Sutherland's) experience had agreed with that; the condition had always passed off in his cases.

Dr. Porter Parkinson said he had seen that day a man, aged 65 years, who had had a peculiar spasm of the left eyelid and

the left side of the face ever since he was five years of age. The only muscles affected appeared to be the orbicularis oculi, and some small muscles of the left side of the face. The spasm was considerably aggravated when the man became excited. He expressed no opinion as to whether it was a case of habit spasm. He had an acquaintance, a gentleman over seventy, who had always had a similar affection, chiefly involving the muscles which pulled the mouth to one side, which was accompanied by a pressing of the tongue into the cheek. In neither case had there been any cerebral or other symptoms. Thus there were certainly some cases, apparently indistinguishable from those of habit spasm, in which the spasm certainly did not pass off, and a too favourable prognosis should not always be given.

Mr. C. H. Milburn (Hull) suggested the use of hypnotism in the case because he had been told that no treatment had been of avail, and his suggestion was in the nature of a last resource. In course of conversation with the mother, he asked her if the boy had had twitchings or movements of the feet. She replied that she had not noticed any ; but immediately the question was asked the boy shuffled his feet. After an interval he asked the mother if there had been movements of the hands. She replied that none had been noticed, but with the question the boy twitched both hands. After five minutes he asked the mother whether there had been movements of the shoulders. She replied she had not noticed any, but immediately the boy twitched both shoulders. That seemed to show that suggestion had an influence in producing movements, therefore probably counteracting suggestion might have a good effect.

Dr. Robert Hutchison, in reply, said he could scarcely share Dr. Sutherland's cheerful view of the future of the case. Every one must know a number of people who grew into adult life still retaining some degree of habit spasm. He would not be surprised if that were so in the present case. He had tried ordinary moral suasion and suggestion, which for a time seemed to do good, but recently the condition had again become worse.

3. A CASE OF BROMIDE RASH.

By ROBERT HUTCHISON, M.D.

A case of bromide rash in a child, aged 2 years, who had been treated with bromide (doses unknown) for two months

for fits. The rash consisted of discrete spots, some of them of large size, on the face and extensor aspects of the arms and back. The spots were not pustular but of the fleshy sort usually met with in young children.

(*Exhibited October the 21st*, 1904.)

Discussion.

Dr. F. J. Poynton thought that often there was no proportion between the dose of bromide given and the facility with which the rash appeared in children. The rash not only did not disappear on the discontinuance of the drug, but continued to come out for some little while. He agreed that the most usual form of the eruption was one like that in the case shown, and had noticed that urticarial and other spots in children seemed to take on a hard and then fleshy form.

Dr. F. Parkes Weber asked whether the kidneys were abnormal in the case, because he believed that persons with kidney disease had a tendency to a "mycotic" form of eruption when bromides or iodides were given.

Dr. Robert Hutchison, in reply, said there was no reason to suppose that the patient had renal disease, but no examination of urine had been made to ascertain that. He would look into the matter.

4. CONGENITAL TROPHIC ŒDEMA.

By OTTO GRÜNBAUM, M.D.

During the last few months three cases of congenital trophic œdema have come under my observation.

Case 1.—T. R—, a male child, aged 3 months, was brought to the Belgrave Hospital for Children because the mother had noticed that the feet were swollen. The parents are healthy and have a child, aged 2 years, which is to all appearances normal. There is no history of abnormal labour, nor any suggestion that the cause of the deformity is due to maternal impression. I remember

seeing a case some years ago which was ascribed by the mother to a fright from an elephant during the period of gestation! The feet alone are affected at the present time; the œdema is symmetrical. There are constrictions at each of the joints of the toes and at the ankles. The child is microcephalic, the circumference of the head being 27·5 centimetres.

Case 2.—The mother of the second child tells me that her mother had swollen legs "during all her life," but there seems to be some doubt whether the œdema was congenital. The father is a painter, but has never suffered from any signs of lead-poisoning nor from any illness whatever. The mother says that her feet swell towards evening, but are normal in the mornings. There are three children—a boy, aged 6 years, who is normal; a girl, aged 5 years; and a baby, aged 6 months; these two latter are affected with œdema of the feet and legs.

M. H— is an intelligent, well-nourished girl with well-marked œdema, and on her I have had an opportunity of making a few observations. The feet, legs, and lower half of the thighs are swollen, the two sides are symmetrical. That this œdema is considerable may be seen by comparing the measurements of her legs with those of a normal child of the same age and build:

	M. H.	Normal child.
Circumference of foot over instep	17·5 cm.	13·7 cm.
Around ankle	17·5 cm.	13·7 cm.
Around calf	20·6 cm.	20·0 cm.
Around knee	22·7 cm.	21·2 cm.

It is evident that the greatest œdema is of the foot and ankle. The toes are very swollen and constrictions mark the joints. The œdema is hard and only pits upon prolonged pressure. The legs are not tender, nor do they ache. Although the mother asserts that the size of the legs is just the same when the child is taken out of bed as when she is put to bed in the evening, nevertheless

posture does make some slight difference in the limbs, but the difference in measurement is insignificant in comparison with the alteration in the consistency of the œdema. The legs are considerably harder after having been dependent for some time. The application of a rubber bandage to them diminishes the circumferential measurement by 5 to 7 mm.

The colour of the skin is normal: the temperature of the legs does not suggest any poverty of circulation.

The blood-pressure in the brachial artery is 91 mm. of mercury. This is the maximum, measured by the obliterative method with C. J. Martin's modification of the Riva Rocci sphygmometer.

It is no easy matter to decide whether the skin of the legs is thickened, for the œdema of the subcutaneous tissue leads one to that conclusion upon the first examination; after prolonged massage I was unable to satisfy myself that there was any permanent pathological alteration of the skin.

Sensation to light touch, pressure, heat, cold, and pain was normal.

A detailed examination of the blood did not seem to me to be devoid of interest, since I thought that it might possibly assist me to decide whether the œdema is due to dilated lymphatics or to excess of lymph in the connective-tissue cells. Blood was taken from a finger and from an œdematous foot before and after massage. Care was taken to make a sufficiently deep incision with a small lancet to lead to a free flow of blood without manipulation. The blood from the finger was found to contain

Red cells . .	5,500,000 per cubic mm.
White cells . .	7600 „ „ „
Hæmoglobin . .	76 per cent.

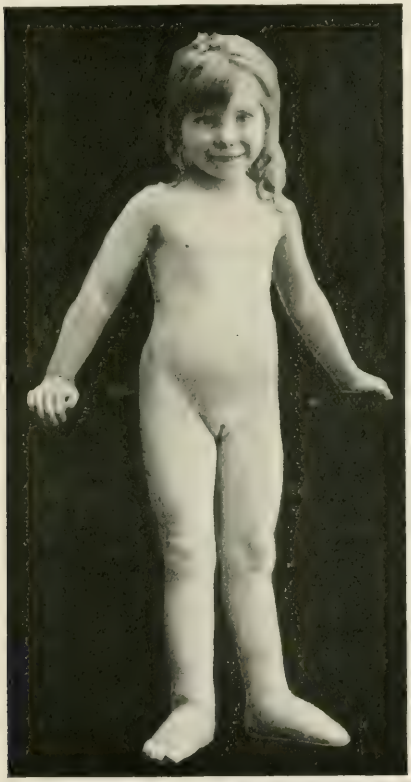
(Haldane's modification of Gower's hæmoglobinometer). Blood from an œdematous foot contained:

Red cells . .	4,250,000 per cubic mm.
White cells . .	6600 „ „ „
Hæmoglobin . .	70 per cent.

After five minutes' vigorous massage of the foot blood withdrawn from it contained :

Red cells	. .	3,240,000 per cubic mm.
White cells	. .	6000 „ „ „

Fig. 1.



The last specimen of blood coagulated in 75 seconds at 22° C. (Wright's coagulometer)—*i.e.* considerably quicker than normal. Massage of normal tissues usually leads to a similar result to the one recorded above, but not to so great an extent. On pricking the skin of an area which has been quite recently massaged, tissue lymph pours out with the blood and dilutes it so that the number of cells

per cubic millimetre is decreased. The fact that the dilution in this case seems to have been greater than normal suggests that the œdema is due to tissue lymph and not to dilated lymphatics, because these would have been partially emptied by massage. The comparison of differential counts of white cells in the blood taken from the finger and the foot corroborates this view:

	Finger.	Foot.
Polynuclear .	49·0 per cent.	49·3 per cent.
Lymphocytes .	40·3 „	40·7 „
Hyaline cells .	4·0 „	3·4 „
Eosinophile .	6·5 „	6·4 „
Basophile . .	0·2 „	0·2 „

(Over 500 cells were classified.)

The similarity of these counts makes it evident that the diluting fluid either contained white cells in the same proportion as the blood or was free from white cells. The fact that the number of white cells was less in the blood taken from the foot than in that taken from the hand makes it certain that the diluting fluid was free from white cells—in other words, that it was tissue lymph and not lymph from lymphatics. The percentage of polynuclear cells is very low for a well-nourished child of five years. The eosinophilia is suggestive of a parasitic affection. Filaria were not seen in any of the blood-films; this, of course, does not prove their absence. It is quite within the limits of possibility that the increase of eosinophile cells is due to some intestinal parasite and has no relation to the œdema. The examination of the blood of the other cases illustrates this.

	T. R. (Case 1), aged 3 months.	J. H. (Case 3), aged 6 months.
Polynuclear .	32·4 per cent.	29 per cent.
Lymphocytes .	63·6 „	63 „
Hyaline . .	1·6 „	6 „
Eosinophile .	2·4 „	2 „

I think that the above facts point towards the con-

dition being due to œdema of the connective-tissue cells and not to dilated lymphatics.

Case 3.—J. H—, a male child, aged 6 months, was admitted into the Belgrave Hospital under Mr. Jaffrey, whom I have to thank for his kindness in allowing me to describe the case. He presents a very similar condition of feet and legs to his sister (Case 2). The feet and legs are swollen, but the feet to a much greater extent than the legs. The skin of the legs is thickened. The skin around the joints of the toes constricts them into three almost spherical masses; the œdema of the terminal phalanges is so great that the plane of the toes is tilted to almost a right angle to the long axis of the toes. The soles of the feet are affected as well as the dorsa; the heels appear to be normal. The measurement around the instep is 13·1 cm., about 1·3 cm. more than the same measurement of a child of the same age.

Since I have three cases only to add to the list it would be out of place for me to give a detailed summary of the numerous instances of this condition which have already been recorded. It may, however, be of interest to see how many varieties of this condition have been described and to note how they differ one from another. Hereditary angeio-neurotic œdema was described by Osler in 1888, but this is quite a different disease from the one under discussion, for the œdema is not persistent and affects different parts at different times. It is, as is well known, associated with other neurotic symptoms, not rarely with intense abdominal neuralgia.

Milroy,* in 1892, wrote a paper on "An Undescribed Variety of Hereditary Œdema," in which he referred to twenty-two individuals in a family of ninety-seven affected with this complaint. The above cases I believe to be similar to those described by Milroy. Meige† recorded trophic œdema in two sisters; in one of these the disease

* 'New York Medical Journal,' 1892.

† 'Nouvelle Iconographie de la Salpêtrière.'

was unilateral. These cases were not congenital. Debove* has termed a condition in which the œdema does not occur *en masse*, but which affects thighs or feet and not toes, as segmental œdema. In one of his cases the œdema began sharply at the gluteal fold and ceased at the popliteal space. There was not any thickening of the skin. Feindel† portrays two sisters affected with œdema in which the disease was asymmetrical and progressive. Osler‡ has described cases of sclerodermia accompanied by œdema, but in these the sclerodermia seemed to be the primary affection. Rolleston§ described a family in which persistent œdema was a marked feature; in his cases the œdema was distinctly diminished by raising the legs; the skin was not thickened.

The conditions which must be recognised are: (1) Congenital hereditary œdema—(*a*) symmetrical, (*b*) asymmetrical; (2) trophic œdema developing in later life; (3) persistent hereditary postural œdema; (4) segmental œdema; (5) sclerodermia with œdema.

Discussion.

Mr. A. H. Tubby said he could not throw much light on the condition, but wished to refer to a certain class of cases of hypertrophy of the subcutaneous tissue which he observed, noted, and tabulated, whilst out-patient surgeon at the Evelina Hospital, and brought before the Edinburgh meeting of the British Medical Association some years ago. Very early in rickets, even before the bone lesions manifested themselves, it was very common to find hypertrophy of the pad of subcutaneous tissue on the dorsum of the foot, and one sometimes saw it extending up the legs, and in many cases it was well marked on the dorsal surface of the hand. He watched such patients for a long time, and found that as the rickets improved, and after the bone lesions had subsided, those pads disappeared. In that disease he believed every tissue of the body was affected in some degree, but the incidence of the affection on the various tissues differed —sometimes the blood-vessels were seriously involved, in others

* 'Presse Medicale,' 1902.

† 'Gaz. Heb. de Med. et Chir.,' 1902.

‡ 'Journal of Cutaneous and Genito-Urinary Diseases,' 1898.

§ 'Lancet,' 1902.

the bones, and in others the connective tissue. There was another form of hypertrophy, truly congenital, which he did not believe Dr. Grünbaum's cases had any connection with, namely, those curious cases of fatty hypertrophy commencing in the feet, which ran a curious but definite clinical course. He had observed six such cases. The one which impressed his mind was that in which he was called in in consultation with four other surgeons and the general medical attendant. The history of those cases, and of that one in particular, was striking. The child was born with congenital fatty hypertrophy of the toes, in consequence of which one or two toes were removed. The fatty hypertrophy recurred, but not in direct continuity with the original injury. It came as discrete outgrowing lobules which subsequently coalesced, and continued its growth some distance up her leg. In its mode of growth and tendency to recurrence this so-called congenital lipoma approached closely to the malignant disease. In one case he dissected a mass from the sole of the foot, and examined the tissue, and the appearances were very much like sarcoma—sarcoma in interstitial connective tissue. In this case the whole fatty growth reappeared in a year, and eventually he had to amputate. He mentioned those scattered facts to show that there was a wide field of investigation in connection with congenital hypertrophy of the skin and subcutaneous tissue in children.

Dr. F. Parkes Weber mentioned that there was a rare but almost identical œdema, not congenital,* and not of a family (hereditary) character, which might come on at any time of life, and led to a chronic thickening of the affected extremity. This was one form of "sporadic elephantiasis," an "elephantiasis glabra," because it was without the very rough or warty skin which characterised most cases of sporadic elephantiasis. Therefore Dr. Weber did not think rickets could have very much to do with the origin of the cases in children now shown. It might be objected that the cases which occurred considerably after birth were of a different class to the congenital and hereditary cases, but he did not think so. They looked the same and corresponded practically in every way. The congenital and hereditary chronic trophœdemas were sometimes unilateral, like those occurring long after birth. He had brought a photograph of a case which he believed to be a case of smooth elephantiasis of one leg in a man who had never had erysipelas or any recurrent or chronic inflammation in the extremity and who had never been out of England. He would like to hear Dr. Grünbaum's opinion of the term sometimes

* *Cf.* the chronic trophœdema case of Lannois, 'Presse Méd.,' May the 7th, 1904, p. 291.

used in France, namely, chronic segmentary œdema.* Surely to regard a foot or a limb as a segment was not correct.

Dr. Porter Parkinson thought the question of prognosis in the cases was illustrated by the fact that the mother of a case under his care attended one of the hospitals in London in 1863, with a similar condition of congenital enlargement of the subcutaneous tissues of both limbs below the knee. In spite of treatment, that enlargement gradually increased, and now both legs were of considerable size. Though the feet had not enlarged in the same proportion, the boots she was obliged to wear were a source of trouble to her. In the case of the child, two or three months' bandaging had no effect whatever, except that one could see where the bandage had been. The œdema was hard, and did not pit on pressure. The possibility of cardiac or renal disease was put out of court in the case of the mother by the duration of the condition, and there was no sign of such disease in the child.

Mr. George Pernet said that Dr. Grünbaum's cases were extremely interesting, because the question of hereditary chronic trophœdema had led to a good deal of discussion. In the younger case there was certainly hypertrophy of the skin of the right leg especially, but he had risen in order to draw attention to an instructive paper by Meige, in the 'Nouvelle Iconographie de la Salpêtrière,' † of which he had a reprint, and a perusal of which would probably interest Dr. Grünbaum, and the members of the Society. It contained many illustrations of the condition as it occurred in another group of cases, in which several generations were affected. Meige mentioned an instance of this, recorded by Milroy in 1893, in the 'New York Medical Record,' in which twenty-two individuals spread over six generations were affected. According to Meige, treatment in such cases appeared useless. Meige further pointed out that in his cases, which developed later than was the case in Dr. Grünbaum's, thyroid had proved useless. With regard to congenital elephantiasis he referred to a paper by Moncorvo,‡ of Rio de Janeiro, in which was a picture of a case something like the one shown at the Bristol meeting of the Association by Drs. Bertram Rogers and Fortescue-Brickdale.

Dr. C. O. Hawthorne said that in the paper just referred to there was considerable evidence as to the existence of a distinct group of cases which could be called chronic trophic œdema. He

* Debove ('Presse Méd.,' May the 28th, 1902, p. 510) says that what he has called *segmentary œdema* is a variety of Meige's *trophœdema* (see Meige, 'Presse Méd.,' December 14th, 1898, on "Hereditary Œdematous Dystrophy," and later accounts by Meige).

† 1899.

‡ Reprint. See also 'Annales de Derm. et de Syph.,' 3e série, tome vi.

had had no experience of the disease, but would say that the present cases corresponded with those described in Meige's paper. Whether the term "trophic" was justified or not, he thought it necessary to insist upon some specific distinguishing term, because there were cases of simple hereditary œdema, *i. e.* cases occasionally occurring in young children, who displayed no hypertrophy of the skin or other parts, the limb being simply œdematous, and the œdema going down when the limbs were raised. A description was given of some cases of this order by Dr. Rolleston in the 'Lancet' of September the 20th, 1902.

Dr. Otto Grünbaum, in reply, said he had looked up the literature, and it seemed to him there were three definite conditions—one mentioned as segmental œdema, where it was not necessary for the ends of the limbs to be œdematous, and which had been ascribed to a segment of the cord being affected, and the nerve-supply from that segment being interfered with, and leading to localised œdema. Another form was that to which Dr. Hawthorne referred. In the case of the two sisters mentioned by Dr. Hawthorne, the œdema developed when they were about sixteen years old.

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5. TWO CASES OF POLIO-ENCEPHALITIS ASSOCIATED WITH MEASLES.

By LEONARD GUTHRIE, M.D.

Case 1. *Cerebral diplegic athetosis.*—Dr. Leonard Guthrie showed (by kind permission of Dr. George Ogilvie) a boy, aged 6 years, who developed measles in February, 1904. On the sixth day he was seized with general convulsions and became unconscious. He remained unconscious for seven weeks, speechless, and paralysed on the right side, with loss of sphincter control.

At present he was rapidly regaining speech, though utterance was very indistinct. He did not appear to be mentally deficient, and was beginning to control his sphincters. The optic discs were healthy. There was no trace of hemiplegia, except slight weakness of the lower right side of the face.

He was unable to sit or stand, and showed in marked degree typical athetoid movements of the trunk and all extremities. The movements ceased during sleep, and were increased on attempted voluntary actions. The knee-jerks could not be elicited; there was no ankle clonus; the plantar reflexes were usually negative, sometimes extensor, in type.

Case 2. *Polio-encephalitis (cerebellar) occurring during measles.*—The child, a girl, aged 6 years, had been shown by Dr. J. Anderson Smith at a meeting of the Society held in April of the present year.*

On February, 1904, on the third day of measles she developed cerebral symptoms—vomiting, convulsions, followed by strabismus, photophobia, and retraction of the head. After five days the acute symptoms subsided, but the patient was aphasic, hyperæsthetic generally, and she wasted rapidly. There was no optic neuritis.

A month after onset she was still aphasic. She could not sit up in bed, and moved her limbs in ataxic fashion, and appeared to have general cutaneous anæsthesia. The limbs were slightly rigid, knee-jerks were exaggerated and ankle clonus was present. Loss of sphincter control persisted for two months.

At present the patient was quite intelligent. Speech was restored but was monotonous and scanning. The optic discs were normal. There was slight tremulousness and ataxy of the upper extremities and marked inco-ordination of the lower. She could not stand without support, showing tendency to fall backwards. Staggering was not increased on closure of the eyes. The gait was typical of cerebellar disease. The knee-jerks were exaggerated, ankle clonus was present at times; the plantar response was flexor, but difficult to elicit.

In commenting on the cases, Dr. Guthrie said that both were probably instances of polio-encephalitis during measles. In the boy's case some slight degree of congenital spasticity had doubtless been present, and the encephalitis had involved chiefly the cortical and subcortical motor areas. In

* *Vide* 'Reports,' vol. iv, pp. 177–9.

the girl's case encephalitis had probably affected chiefly the cerebellum.

He drew attention to Dr. F. E. Batten's valuable series of cases illustrating the occurrence of polio-encephalitis in different areas of the brain, and their relationship to anterior polio-myelitis.*

(*Exhibited October the 21st*, 1904.)

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6. INFANTILE PARALYSIS WITH PERMANENT INVOLVEMENT OF THE ABDOMINAL MUSCLES.

By ARTHUR EDMUNDS, M.S.

The child, aged 12½ years, was first brought to Paddington Green Children's Hospital in 1895, when she was 2½ years old.

Previous to this she had had a severe attack of whooping-cough, and a few months before the onset of the paralysis she had had measles.

The paralysis came on suddenly, and was very extensive, the whole four limbs and the muscles of the head and neck being affected. The condition gradually improved until the only affection seemed to be a weakness of the right leg, but a month later the mother noticed a swelling of the abdomen on the right side. She was brought to Paddington Green Children's Hospital, and in November, 1895, was admitted under the care of Dr. Sutherland, to whom I am indebted for notes of the case at this time.

Her condition at this time did not differ very greatly from that now obtaining. There was partial paralysis of the right leg, with wasting and impairment of the circulation; the knee-jerk was absent. There was no affection of sensation or of the sphincters.

The abdomen, however, which did not present any

* 'Lancet,' December the 20th, 1902.

marked peculiarities when the child was at rest, showed a very pronounced ballooning when there was any rise of intra-abdominal pressure—for example, on crying. The ballooned area extended from the costal margin to Poupart's ligament, and from the outer border of the erector spinæ to the middle line. With the exception of slight rickets the general condition of the child was good, and after a course of massage and electrical treatment she was discharged from the hospital, able to walk and distinctly stronger.

The patient then passed from observation for nine years, at the end of which she was again brought to the hospital for the relief of several deformities which had become apparent. Her condition at the present time is as follows: She is a fairly well nourished child of normal intelligence. She is in the fourth standard at school, although, like so many of these cases, her power of resisting disease has been diminished, and she has fibroid phthisis on the left side. Her left pupil is larger than the right, but both react normally.

The improvement of the leg has not made much progress since 1895, but she is able to get about well in spite of frequent falls owing to the limb giving way under her. The knee-jerk is still absent.

The ballooning of the abdominal wall can still be demonstrated, but the extent of this has diminished, there being a patch just below the costal margin and a patch above Poupart's ligament, where recovery has taken place.

The weakened portion of the abdominal wall extends from the outer border of the erector spinæ muscle to the middle line, being bounded above and below by two curved lines with the convexity downwards. These lines meet in the middle line just above the umbilicus, enclosing an area which corresponds roughly to the area supplied by the 8th and 9th dorsal nerves.

The edges of this are sharp, and can be distinctly felt. When the child blows out her cheeks, the affected portion of the abdominal wall bulges out, forming a swelling as

large as a closed fist. This abdominal condition does not seem in any way to affect either the child's health or her comfort.

Faradic excitability is completely lost over the paralysed area, and there is no response even to the constant current.

The right leg is 1½ inches shorter than the left and about an inch less in circumference. Faradic excitability is diminished, but present, being more marked below the knee than above. There is no laxity of the joints.

In the back there is a well-marked scoliosis, which takes the form of a curve in the lumbar region, with the convexity to the right (paralysed) side, and this is accompanied by a well-marked rotation of the bodies of the vertebræ. This rotation can be readily examined through the deficiency in the abdominal wall, and the bodies can be felt projecting well beyond the outer border of the erector spinæ.

There is a long, well-marked compensatory curve in the dorsal region. By appropriate manipulation these curves can be completely obliterated.

The curvature of the spine is mainly due to the shortening of the right leg, although the weakness of the abdominal wall may play some part in its causation, the direction of the curve being that which one would expect from such a condition.

There is a further factor in the spinal curvature which may play a greater part in the future than it does at present, namely, the contraction of the left lung which seems to be taking place; its effect would be to straighten out the dorsal spine.

The child will require some form of knee-cap to prevent her from falling about, as I am not very confident of sufficient improvement taking place to allow her to walk in safety without a support. I have provided her with a temporary splint of plaster of Paris, which seems to be quite satisfactory. She will also need a high boot to bring the pelvis horizontal and remedy the scoliosis, while the abdominal condition will be met by a well-fitting abdominal belt. *(Exhibited October the 21st, 1904.)*

Discussion.

Dr. G. A. Sutherland said he thought the treatment of the case must be regarded as surgical. A point which was of interest from the medical standpoint was the localised bulging in the abdominal wall on the right side, which was still present. When the child was first seen, the whole of that side was paralysed, but under treatment by faradism and galvanism, the greater part recovered nine years ago. There was a localised bulging under the ribs on the right side. If one had not known the history of the case, and it had only just been seen, he thought another possibility would have suggested itself, namely, that it was congenital absence of the muscular wall on that side. The remains of that paralysis on the right side were in the same position as that in which one usually found a congenital absence of muscle in the abdominal wall. It might occur on the right or the left, or on both sides, but it was always underneath the ribs, and nearer to the side than to the middle line. The cause of the congenital lesion was not well understood. One theory was that it was due to the fact that during intra-uterine life the fœtus was fixed so that the knees pressed on the abdomen, and that the continuance of that pressure produced atrophy of all the structures under the skin.

7. DERMOID CYST OF THE AURICLE.

By P. LOCKHART MUMMERY, B.C.

The patient is a boy, aged 8 years. His mother says that he was born with a small lump in front of the upper part of the left ear. This lump gradually increased in size until it was as big as a hazel-nut. It never caused him any pain or inconvenience. Two and a half weeks ago he was knocked down by a bicycle and bruised the left side of his head. Two days later it was noticed that the lump was larger, and also that it was painful.

The boy has no congenital defects, and there is no history of such in the family.

The lump consists of a soft fluctuating cyst about an inch in diameter just under the helix of the left auricle and in front of it. There appears to be a depression in the bone

beneath. The skin is not adherent over the cyst, and is quite normal in appearance except for some bruising. There is no pulsation to be seen in the swelling.

The history makes it seem probable that the cyst is a dermoid; the position is, however, an unusual one for a dermoid. Dermoid cysts have often been met with just in front of the meatus, due to persistent portions of the intermaxillary cleft, but the position of this cyst does not correspond to the line of this cleft, and it seems very improbable that it can have arisen from it.

(*Exhibited October the 21st*, 1904.)

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8. CONGENITAL HEART DISEASE

By EDMUND CAUTLEY, M.D.

The patient exhibited was interesting on account of the great difficulty in making an accurate diagnosis of the defect in the heart and the association of other congenital abnormalities. As in so many similar cases, there was a possibility of the heart defect being dependent on congenital syphilis. The girl, aged 3 years, had come under treatment for bronchitis. She was one of six living children. The mother had lost two children and had had eight miscarriages. One child, aged 16 years, has a "weak heart" due to rheumatism. The patient was of normal size, fair nutrition, but rather anæmic. A blood examination showed 4,500,000 red cells, hæmoglobin 62 per cent. The apex of the heart was felt just outside the nipple line in the fifth interspace. The impulse was a trifle exaggerated. There was no increase in the area of dulness to the right, but a distinct increase upwards on the left side, as high as the second rib, and outwards to the left. A systolic thrill was felt over the second and third left intercostal spaces near the sternum. A long systolic murmur was

heard all over the præcordium, loudest in the same area as the thrill was felt. The murmur was conducted upwards into the neck on both sides, being very much louder and rougher on the left side. Sometimes a short diastolic murmur could be heard in the second and third left intercostal spaces. Sometimes reduplication of the second sound at the left base was noted.

In addition, the child presented paralysis of the left superior rectus muscle, the other orbital muscles being unaffected, and an abnormal artery running down the middle of the back of the left forearm and over the wrist. The pulse was sudden and of good volume. At one time the pulse had been of the typically water-hammer type.

At first Dr. Cautley had suspected the possibility of double aortic disease, but the character of the murmur, its conduction to the left clavicle, and the size of the pulse were opposed to this view. He suggested that there might be a direct communication between the aorta and pulmonary artery above the valves, or that the condition was one of patent ductus arteriosus. He could not accept a diagnosis of pulmonary stenosis by reason of the absence of distinct evidence of hypertrophy of the right ventricle, of cyanosis, and of clubbing. Considering the loudness of the murmur and the thrill, one would have expected more effect on the right side of the heart and on the circulation generally if the case were one of pulmonary atresia or stenosis. Moreover, he thought that the unusual extension of the dulness upwards on the left side was opposed to this view. Of course, it was possible that there was more than one lesion.

(*Exhibited October the* 21*st*, 1904.)

Discussion.

Dr. C. W. Chapman thought it was impossible to say what the condition was. He considered the practical point was the question between pulmonary stenosis and the other lesion which Dr. Cautley suggested. One would think it was pulmonary stenosis until it was found that there was no dilatation on the right side of the heart.

9. IMBECILITY WITH MICROCEPHALY, SPASTICITY AND IRREGULAR TREMORS.

By EDMUND CAUTLEY, M.D.

The child, a female, aged 21 months, was the fourth of five. The labour was an easy one, without instruments, and there was no lividity noted at birth. No history of congenital syphilis was obtained. The mother had not had any miscarriages. She had lost one infant from diarrhœa and another from convulsions, both, no doubt, due to errors in diet. Patient had never had fits. Movements of the head and limbs were noted at about the sixth month of life, and have gradually got worse. The child has never been able to sit up, crawl, walk, or talk, and has very little power of holding up her head.

Her present condition is one of imbecility. She looks healthy, placid, and happy, but has paroxysms of temper, lasting as long as an hour at a time. She swallows fluids badly, and is dirty in her habits. She grasps, but soon drops articles placed in her hands, and does not play with toys. Her head is small, measuring 17½ inches. Irregular lateral and nodding movements of the head are common, somewhat like typical "spasmus nutans." Irregular, jerky, and tremulous movements of the hands and arms, of the trunk and lower limbs, are also seen. These are variable in extent, most marked in the hands and arms, best seen when the child awakes from sleep, or on movement or excitement, and are somewhat athetoid in character. A certain amount of general rigidity pervades the whole trunk and limbs, especially the legs. The knee-jerks are exaggerated. Occasionally very slight lateral nystagmus is seen. The sacral fossa is well marked.

Although the case might be diagnosed as microcephalic idiocy, seeing that the child has a small head and is imbecile, it is probable that there is something more present. The rigidity and exaggerated reflexes suggest the possibility

of some birth palsy, a spastic diplegia of mild type. Dr. Cautley thought that it should be diagnosed as a mild case of spastic paraplegia in a microcephalic idiot. He did not think the microcephaly alone would account for the spasticity, nor did he think that with such a mild degree of spasticity the mental state would be so defective, unless there were another factor concerned. The prognosis was hopeless.

(*Exhibited October the* 21*st,* 1904.)

Discussion.

The Chairman (**Dr. Fletcher Beach**) said he had seen several cases of the kind when he was at Darenth Asylum many years ago, children with small heads with a spastic condition of the limbs, who never practically recovered the use of their limbs, but he had not seen a case in which tremors had been so marked as in the present case. He was inclined to think it was a case of imbecility associated with a spastic condition of the limbs, and such as could be often seen.

Dr. Porter Parkinson asked whether there was any history of prolonged labour, or asphyxia at the time of birth. He saw from time to time a case of microcephalic idiocy, with a good deal of depression on either side of the forehead, and in that case there developed certain nervous phenomena other than purely mental ones, viz. a spastic weakness of both legs, which had been maintained up to the present time, three years since he saw the case. There was in that a distinct history of very prolonged labour, and the child was born in a state of cyanosis, from which it was with very great difficulty revived. Since that time there had been convulsions also ; the symptoms appeared to come on slowly and were progressive.

Dr. Edmund Cautley, in reply, said he might have mentioned that the child was the fourth of five children, and therefore the labour would not be expected to be difficult, and it was not. There was certainly no asphyxia, nor anything in the family history which could be taken to account for the condition. Two of the other children were living and healthy, and the other two died at the age of four months, one of marasmus and the other of convulsions, the marasmus no doubt being due to bad feeding.

10. A CASE OF CONGENITAL BILATERAL AND SYMMETRICAL DISLOCATION OF THE CRYSTALLINE LENSES.

By N. BISHOP HARMAN, M.B.

F. H—, male, aged 14 years, small and frail for the age. Father dead from consumption; mother alive and well; has one brother deaf, but well sighted, and one healthy and well-sighted sister. The boy was brought up at Saint —— Home, in Liverpool. He has been taught to read a little, but has received no instruction in handicraft, and has, as far as can be ascertained, never before been taken to have his eyes examined by a surgeon, with a result that he now ekes out a poor existence by sweeping an office. The occurrence of such examples of gross neglect of children in the care of pretentious ecclesiastical and philanthropic "homes" makes a strong argument for the proper medical supervision of the children in all such institutions, in the manner which has been so well worked out by the old School Board for London, and continued by the London County Council.

The youth was seen first on October the 4th of this year. It was at once noticed that both lenses were dislocated. He presents no other abnormalities than those of the eyes; the teeth are excellent both in quality and condition.

Each lens is displaced inwards and upwards in the direction of the root of the nose; their exposed edges are well rounded and slightly opaque. There is no trace of any suspensory ligament for fully one half of the perimetry of the lens, and from the position of the lens it may be judged that the ligament is absent for two thirds of its normal extent. There is in each eye a clear crescent-shaped pupillary area below and to the outer part; through this area the fundus can be seen; it is normal in each eye. The irides are abnormal in that they cannot be dilated by midriatics.

When a strong light is thrown upon the pupil in the dark room there is a contraction to its ordinary daylight

size. Each iris is very tremulous, and the peculiarly heavy movement of the upper and inner quadrant rather suggests that the lens sways backwards and forwards behind the iris.

The right eye fixes, and has vision at a distance of three inches of Jg. 6 with some difficulty. Retinoscopy through the clear crescentic portion of the pupil gives a refraction of + 9D. With this glass the boy read at once 6-18, and one letter of 6-12; with + 13 D. he read Jg. 4 at 8 in., and on a second attempt some of Jg. 2. It seems probable that his visual acuity will improve with a regular use of the glasses.

The left eye diverges, and has no quantitative vision. Retinoscopy gives a refraction of + 9 D., but a glass does not aid vision. The eye is probably amblyopic from suppression of vision by disuse; his habit of holding printed matter very close to the right eye completely precludes the possibility of the simultaneous use of the left eye.

It has been shown by the researches of Mr. Treacher Collins that the lens in early fœtal life has a size which, in relation to the rest of the globe, is greater than that which it attains at any other time; it is so large that it is in close contact with the ciliary body. During this period its equator becomes adherent to the ciliary region, and as the globe grows at a greater rate than the lens, the links of connection between lens and ciliary region become drawn out to form the slender strands of the suspensory ligament. We must conceive that in the case of this boy for some reason these primitive adhesions did not form around the whole lens, but only inwards and upwards; the condition, then, is not a dislocation in the proper sense of the word, but a failure of union. The defect does not appear to be in the lens, for the epiblast from which the lens arises, in common with the scarf-skin, hair, and teeth, is normal; rather it would appear to be in the irido-ciliary region, for it has been noted that the iris of each eye was defective in that it could not be dilated. Probably the retractor muscles are absent, or only developed in a slight degree.

The treatment of these cases is rarely so easy or satisfactory as in this boy. He has a clear pupillary opening, and can be treated as aphasic and given ordinary cataract glasses with excellent results. When the badly placed lens obstructs the pupil it is usual to remove it by needling; the practice is not as simple as in the case of needling an ordinarily suspended lens; the displaced lens is very tolerant of injury, and does not ripen with facility after its capsule has been apparently well opened up by the needling. I believe the difficulty arises from the lack of tension on the capsule of these lenses. It is not pulled upon equally from all directions at its equator, so that a prick, or even a cut does not cause the capsule to retract and expose the lens matter. The condition can be illustrated by the different effects of pushing a needle into a piece of fine kid. If the kid is loosely held in the fingers, the prick leaves a hole not easily found; if the kid be tightly stretched upon a "drum," the prick stretches out into a wide hole. I am inclined to think that it would be much better to open the anterior chamber and boldly remove the capsule of the lens by seizing it with Couper's capsule forceps, steadying the lens if necessary with a needle. The operation should entail no added risk.

(*Exhibited October the 21st*, 1904).

11. A CASE OF CEREBRAL DEGENERATION WITH FUNDUS CHANGES.

By SYDNEY STEPHENSON, C.M.

The following case appears to be an example of a condition recently described in children, where cerebral degeneracy is associated with pallor of the optic discs and slight pigmentary changes in the central region of the fundus oculi.

The facts are as follows:

Albert B—, aged 7½ years, was brought to the ophthalmic department of the North-Eastern Hospital for Children, on

May the 19th, 1904, with the statement that during the past year his sight had failed and he had become dull, stupid, and "nervous." He had formerly been a bright and healthy lad.

Family history.—The mother and father are not connected by ties of blood. They have been married for about nine years, and have had no offspring except the patient. There have been neither miscarriages nor still-births.

Personal history.—The boy suffered from bronchitis, German measles, and influenza at $4\frac{1}{2}$ years, followed by measles six months later and by chicken pox six months after that. At $6\frac{1}{2}$ years he developed mumps.

Upon examination.—This intensely "nervous" lad seems to have little direct vision, since he walks "crab-wise" and never looks straight at an object held up before him. Usually, indeed, he stares in a fixed and sightless way. The pupils are of medium size, equal, and have but little direct response to light. The movements of the eyeballs are limited in all directions, save downwards. The optic discs are rather pallid; the retinal vessels are of fair size. The "gunpowdery" appearance of both fundi is abnormally pronounced, and becomes accentuated in and about the yellow spot region. No personal evidence of syphilis could be obtained. Dr. George Carpenter, whom I asked to see the patient, reported that the plantar reflex showed an extensor character, and that all the other reflexes were present.

Progress and treatment.—Potassium iodide, grs. 2 *per diem.*

July the 14th, 1904.—The lad now remembers nothing properly, and has become quite uneducable. The pupils, especially the left one, sluggish. Movements of eyeballs limited in all directions, particularly upward.

August the 11th, 1904.—Pupils equal, and more active than they were. Upward movement of eyeballs almost absent; lateral movements restricted; downward movement good. R.V. = $\frac{1}{60}$ and No. 19 Jaeger; L.V. = $\frac{1}{60}$ and No. 19 Jaeger. Central vision, if not absent altogether, is certainly most defective. Dr. James Taylor, who did me the favour of examining the patient, reported that there were "no obvious signs of structural nervous disease."

September the 22nd, 1904.—Patient stated to be brighter in himself.

October the 20th, 1904.—The boy recognises with either eye such familiar objects as matches, pennies, and brushes at two feet distance. He reads (with either eye) No. 14 on the Jaeger hand-type. That central vision is absent may be inferred from the fact that he never looks straight at an object, but to right or left of it. The downward movement of the eyes is good, but the others are performed most imperfectly. Pupils equal, with fair reaction to light. Slight but distinct hippus. The optic discs are somewhat whiter than normal, especially as regards their outer side. The slight pigmentary changes of the fundus remain as noted.

Remarks.—The symptoms in this case appear to me to point to widely-spread degenerative changes affecting both the cortex and the base of the boy's brain. Cases of a somewhat similar kind have been reported by :

(1) Batten, Rayner, D.—'Ophth. Soc. Trans.,' vol. xvii, 1897.

(2) Still and Gunn, D.—*Ibid.*, vol. xx, 1900, p. 108.

(3) Batten, F. E.—*Ibid.*, vol. xxiii, 1903.

(4) Hirschberg, J.—'Centralbl. f. prak. Augenheilkunde,' January, 1904.

(5) Mayou, S.—'Ophth. Soc. Trans.,' vol. xxiv, 1904.

(6) Hutchison, R.—Society for the Study of Disease in Children, February the 19th, 1904.

(*Exhibited October the 21st*, 1904.)

Discussion.

The Chairman (**Dr. Fletcher Beach**) said he would like to know if the child was dull, because there was a class of case which had been described as amaurotic idiocy, and he did not know whether the present case was sufficiently dull to be classed as an idiot. Some cases had been described by Risien Russell before the Royal Medical and Chirurgical Society with very much the kind of symptoms mentioned, and all of them ended fatally. Possibly Mr. Stephenson's case belonged to the same category.

Mr. Bishop Harman said he was interested in these cases, as he had seen some examples amongst the children of the London County Council Blind Schools. There were in these

schools over fifty children blind from optic atrophy, and he believed that amongst them there was every possible gradation of fundus change, from the slight delicate damage such as in the case shown by Mr. Stephenson to the most complete atrophy with gross choroidal changes. He had found it difficult to draw a hard and fast line between these particular cases and those freely acknowledged to be disseminated choroiditis of syphilitic origin. One circumstance had impressed this upon him greatly: A couple of years ago he saw two pretty little girls of good and apparently healthy parentage; both were blind centrally, only some peripheral perception remained. The elder had been previously seen by a distinguished ophthalmic surgeon who had diagnosed disseminated choroiditis of hereditary syphilis, and now the second had been born and gone blind with just these slight changes described. He was not convinced that these cases should be grouped absolutely alone in one solitary patch, as Mr. Stephenson and others suggested, because they shaded off into those conditions no one would hesitate to call disseminated choroiditis.

Mr. Sydney Stephenson, in reply to the Chairman, said the child was very dull. The two conditions resembled one another in that they were family affections; but the age of the patients and the character of the ophthalmoscopic changes and, so far as was known, the termination of the cases, differed. He passed round a picture which was published by Dr. Frederick E. Batten. It was interesting to hear Mr. Harman's experience, as he devoted attention to the eyes of children, but he could not agree when he suggested that he (Mr. Stephenson) should attempt to form them into "a little isolated patch" of cases. The cases might shade off into syphilitic choroiditis, but even if they did—and he was not sure about that—he was not aware that syphilitic choroiditis was associated with mental failure, ophthalmoplegia, and the other symptoms which characterised the patient he had shown that evening.

12. A CASE OF TRICHOTILLOMANIA.*

By GEORGE PERNET.

Mr. George Pernet showed a photograph of this condition, for which he was indebted to Dr. L. Raynaud, of Algiers. The patient was a lad, aged 11 years, who was in

* τιλλειν, to pluck out, etc.

the habit of pulling out the hairs of his scalp and sucking them. There was a morbid sexual element in the case. The patient was under the care of the late Professor Gemy, of Algiers.

Fig. 2.

Discussion.

The Chairman (**Dr. Fletcher Beach**) said he had seen many cases apparently similar to that in the photograph, and there were a certain number of such children who pulled out their hair. There were some cases in Darenth Asylum who pulled out almost every hair until they were practically bald. He had not previously heard the name now applied, but the photograph showed the case to be evidently a degenerate.

13. A CASE OF HERPES ZOSTER EXTENDING TO THE FINGER-TIPS.

By GEORGE CARPENTER, M.D.

Herpes zoster extending to the fingers is rare in any event, especially so in children. The accompanying photo-

Fig. 3.

Fig. 4.

graphs well illustrate the condition which I observed some years ago in a child under my care at the Evelina Hospital for Children. The eruption affected an extensive cutaneous area, viz. the front and back of the upper part of the chest, the arm and forearm in front and behind, the back

of the arm, also the palm of the hand and the little, ring, and middle fingers, with a small patch at the root of the little finger on the back of the hand. The nerve roots affected were the eighth cervical and the upper three dorsal nerves. The girl made an uninterrupted recovery and nothing worthy of note occurred in the case.

(*Exhibited October the 21st*, 1905.)

Discussion.

Mr. George Pernet said the case was one of great interest, because it was extremely rare to find Herpes zoster affecting the fingers, but recently he had seen a woman with Herpes zoster of the left leg who had several vesicles and one bulla on the sole of the foot.

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14. ON AN ILL-DEVELOPED UPPER LATERAL INCISOR TOOTH AS A FORERUNNER OF HARELIP OR CLEFT PALATE.

By R. CLEMENT LUCAS, B.S.

There is a tendency and often a desire to represent a congenital deformity as something which has suddenly developed *de novo* and as a defect which is to be explained by some accidental occurrence during pregnancy or labour. An enormous amount of ingenuity is in many cases expended by the parents, relations, friends, and even the family doctor, to place the defect before the world as a mere accident that would never have occurred if such and such a sight had not by chance been encountered by the mother during her pregnancy, an incident that occurred probably long after the establishment of the defect *in utero*, and entirely forgotten till raked up to explain away the unfortunate deformity in the child. Superstitions die hard even in a highly civilised and generally intelligent community, and blame is an imputation which every one desires to transfer from himself to his neighbour; or should no one be found conveniently placed to bear it, then a spiritual influence that cannot be questioned is

usually called in to account for what natural causes may still be able to explain.

I have attempted to show that in the case of harelip and cleft palate there may have been through generations an unnoticed defect in the natural line of junction between the incisor bone and maxilla. Then the marriage with another who by chance has inherited through some ancestor a weakness of development in the same part, or possibly, simply as the result of deprivation of proper nutriment during the developing period, a well-marked distinct and obvious deformity noticeable by all may become developed.

Besides the ingrained tendency to adhere for all time to superstitions which fancy favours, however reason rebels, there is another very human faculty, which is to accept nothing new from any one with whom one has the slightest acquaintance, unless perchance it has first been published in a foreign language as the observation of some distinguished foreign professor. Thus, sound observations often lie dormant for years till some honest worker in the same field unearths what might have prevented many fallacies had it been tested, and proved or discarded, shortly after the observation had been made. From this neglected heap I dig up the following observations published sixteen years ago.

In the Clinical Society's 'Transactions' for the year 1888, page 64, I published a short paper "On the Congenital Absence of an Upper Lateral Incisor Tooth as a Forerunner of Harelip and Cleft Palate." There were three observations bearing upon this relation in the paper referred to. In the first a gentleman who, as a boy, was noticed to have an absence of the left upper lateral incisor, had an only daughter who presented precisely the same defect on the same side. As this only child has never married there is no chance of proving the relation of the defect to harelip and cleft palate in that family. It proves, however, that the congenital absence of an upper lateral incisor tooth is a defect which may be transmitted, and that the transmission may be through the opposite sex.

The second case was brought among my out-patients at the Evelina Hospital in 1883. An infant suffering from harelip on the right of the median line was brought by a mother who had congenital absence of the right upper lateral incisor tooth. The mother was twenty-five years of age, had been married seven years, and this was her fifth child. There was an interval of one year and eight months between this child and the one before, and she had continued to suckle until she fell pregnant again. The mother's lip was perfectly normal. There was no notch or scar upon it, but her right upper lateral incisor was known to have been absent, and it had never been extracted. The other teeth were well formed. The child had also double inguinal hernia and an umbilical one. It would seem that the exhaustion of the mother by repeated pregnancies and prolonged suckling was a factor in determining the appearance of the deformity.

The third case was that of a child with harelip and cleft palate on the left side, whose mother had absence of the left upper lateral incisor tooth. She was twenty-three years of age, and this was her first child. They came under observation in April, 1887. The mother was one of a large family, none of whom had shown any deformity. She was a pale, delicate-looking woman. A sister and an aunt had died of consumption. She knew of no defect in her husband's family. Her lip was perfect. She was positive that no tooth had ever been extracted from the front of her mouth, though she had lost one or two molars.

From these observations I concluded that *the absence of an upper lateral incisor tooth resulting from an arrest of development is to be regarded as a malformation closely related to harelip and cleft palate, and capable of transmitting each or both of these deformities to a succeeding generation.*

I have now to record two further observations which show that a lesser deformity than congenital absence of the upper lateral incisor tooth, and one more likely to be overlooked, may indicate the possible occurrence of harelip

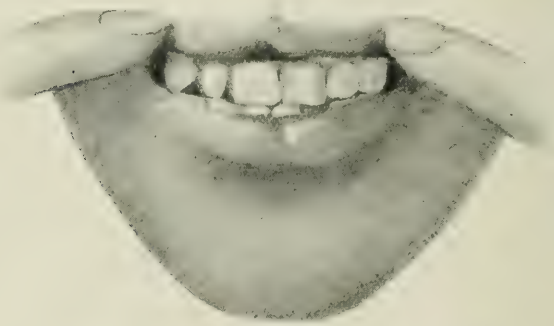
or cleft palate in a subsequent generation. These observations, which I mentioned in the Wightman Lecture, go to show that a defective development in an upper lateral incisor is a danger signal in reference to these deformities which should not pass unnoticed.

CASE I.

Right-sided harelip and complete cleft palate in the first child of a mother who had an imperfectly developed right upper lateral incisor tooth, and who had been operated on for harelip in childhood.

E. R—, aged 3 months, was first admitted under the late Mr. Durham's care into Guy's Hospital on October

FIG. 5.



the 8th, 1892, and was discharged without operation on January the 27th, 1893.

She was readmitted under Mr. Clement Lucas's care in Lydia Ward, on July the 26th, 1893, and the case was reported by Mr. F. W. Rowland.

The mother showed the scar of an operation in childhood for harelip, but the defect had not extended to the palate. The teeth were all well developed except the right upper lateral incisor, which was narrow and pointed towards the end. A painting was made of this condition by the artist to the hospital.

The infant, a girl, presented an oblique fissure through the upper lip to the right of the median line extending into the nostril. On the left side of the fissure the premaxillary bone projected prominently forward, and to the right. The cleft extended backwards along the roof of the mouth through the alveolus and hard and soft palate. The fissure in the lip was about a quarter of an inch in width, whilst that in the palate was about half an inch. The child was extremely thin.

On August the 18th the harelip was operated on, and on August the 24th, complete union having taken place, the child was discharged.

Case II.

Harelip in an infant whose mother had a perfect lip, but an ill-developed upper lateral incisor on the same side as the child's harelip.

W. J—, aged 6 weeks, a boy, was admitted into Martha Ward, Guy's Hospital, on June the 10th, 1903, and discharged on June the 20th as he was not in a fit state for operation.

He was readmitted on August the 17th, 1903, and discharged on September the 18th after operation.

The case was reported by Mr. F. J. H. Wood. The patient was the first child of young parents. The left side of his upper lip presented a cleft which extended up into the left nostril. The left nostril was much wider than the right. The mother knew of no deformity of the kind either in her husband's or in her own family. Her own upper lip showed no defect, but Mr. Lucas, on examining her teeth, pointed out that she had a remarkable narrow ill-developed upper left lateral incisor tooth which he considered was related to the deformity in the child. The mother's teeth were photographed, and a sketch was also made showing the relative size of the defective tooth to the others, which were all large, well-developed teeth.

September the 8th.—The harelip was operated on and sealed iodoform dressings were applied over.

September the 11th.—The sutures were removed.

September the 18th.—He was discharged cured.

Fig. 6.

When I read my paper sixteen years ago, "On the Absence of the Upper Lateral Incisor as an Indication of the possible Occurrence of Harelip and Cleft Palate in a Succeeding Generation," some dental surgeons present declared that the lack of eruption of certain teeth of the

second set was a matter of common observation, and that no clinical importance need attach to it. But what I wish to again insist upon and to maintain is, that with a weakened development in the line of junction between the pre-maxillary and the superior maxillary bones the lateral incisor tooth becomes involved in the defect, and may either fail to be cut or, if erupted, may show a defective development that is a danger-signal as to results which may happen in subsequent generations. It will be observed that the obvious secondary deformity may show itself in

FIG. 7.

the soft structures alone as a harelip, or both palate and lip may be involved in the next generation.

So far, I have no observations on the condition of the milk teeth in these cases, but I think it highly probable that the primary teeth may indicate the defect as well as the second set.

It is seldom that one has an opportunity of examining the father's teeth, but, as in the first observation made, the absence of a lateral incisor was transmitted to a daughter, it shows the necessity of examining the teeth of both parents to obtain evidence of a prior defect.

Further, it must not be expected that every child of a

person showing defective development or absence of an upper lateral incisor should inherit either the same or an increased deformity; and although most of those recorded were first children born, in one case four normal children were born before a deformed child appeared.

(*Read October the 21st*, 1904.)

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15. TWO CASES OF CONGENITAL DIAPHRAGMATIC HERNIA.*

By SIDNEY GILFORD, M.B.

The two following cases recently admitted into the Paddington Green Children's Hospital are of interest, as they represent a rare variety of congenital diaphragmatic hernia.

Case 1.—A female child, aged 4 months, was admitted on December the 3rd, 1903, in a moribund condition, under the care of Dr. Guthrie. The child was unconscious, with sunken fontanelle and internal strabismus of the left eye. Her temperature was 103·8° F. The pulse was imperceptible at the wrist, and the respiration was slow and sighing. There was retraction of the abdomen, but no *tâche cérébrale*. Death took place fourteen hours after admission. She was sick six times, each time bringing up dark brown vomit. It was thought probable that she was suffering from cerebral thrombosis.

Post mortem.—Body well nourished. On opening the abdomen the liver was found to extend almost down to the symphysis pubis, and the gall-bladder, which was in the middle line, was in contact with it. The liver was rotated so that the upper surface looked forward.

On opening the chest it was found that the whole of the lower and external portion of the right side was occupied by the large and small intestines, the right lung being

* "Congenital Lesions of the Diaphragm and Respiratory Insufficiency in New-born Children," by Ambrogio Mori, M.D., 'The British Journal of Children's Diseases,' pp. 115–21, March, 1905.

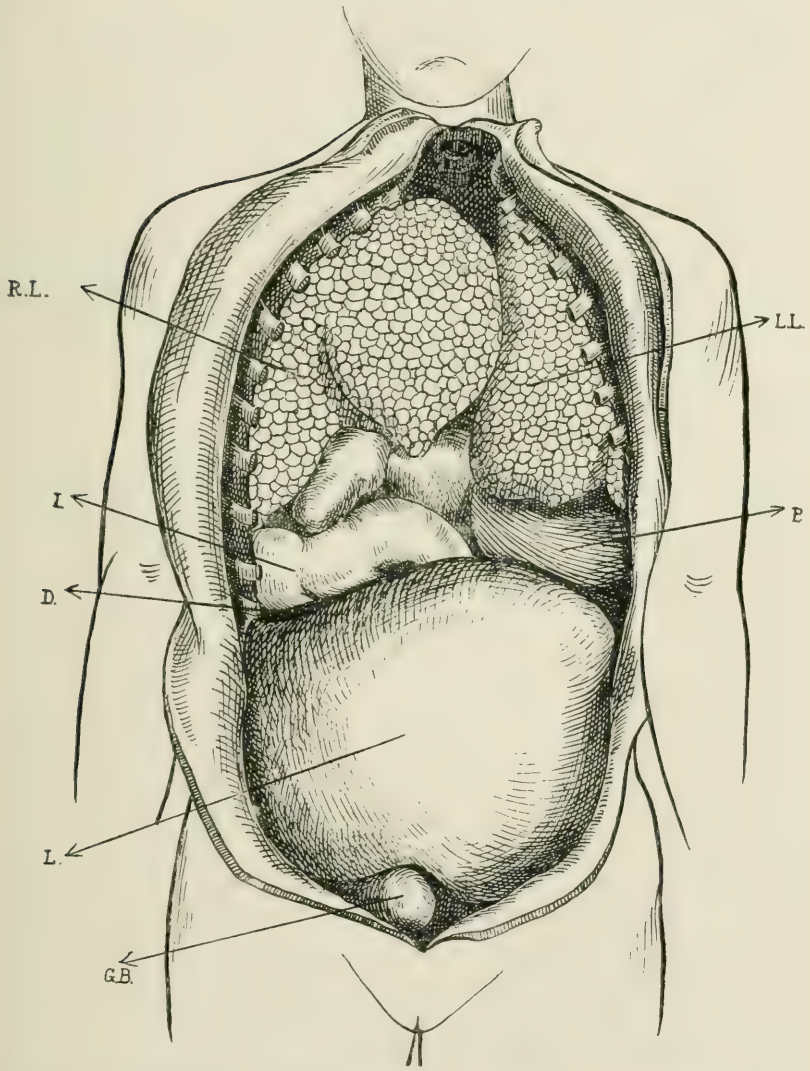

FIG. 8.—CASE 1.—R.L. Right lung. L.L. Left lung. I. Intestine. P. Pericardium. D. Diaphragm. L. Liver. G.B. Gall-bladder.

displaced upward and towards the middle line. The anterior mediastinum and the heart were displaced to the left, the right side of the heart being entirely to the left of the left border of the sternum. The diaphragm was pushed down and flattened. The stomach occupied its usual position, but the duodenum, which had a mesentery, passed upwards and to the right and disappeared behind the liver. The large intestine passed obliquely across the abdomen to reach the rectum. The abdominal contents, which had passed through the diaphragm into the right side of the chest, were the third part of the duodenum, the jejunum, the ileum, the ascending and transverse colon, and part of the pancreas. There was no sign of strangulation; and fæces were present in the lower part of the intestine and appeared normal.

The opening in the diaphragm was lying to the right of the middle line external to the crus. Its circumference was about two inches, and the peritoneum was continuous with the parietal layer of the pleura. All the organs appeared healthy, and no cause of death was found.

Case 2.—A male child, aged 4 months, was admitted on May the 24th, 1904, under the care of Dr. Sutherland.

History.—He was well until six weeks ago, when he had what his mother described as a fit, accompanied by cough and cyanosis. Similar attacks occurred frequently, and finally he was sent in by a doctor who thought there was fluid in the right pleural cavity. He had been fed upon breast-milk only, and had always taken it well. The bowels were usually regular, but he frequently had abdominal pain, and occasionally suffered from constipation.

On admission.—There was extreme cyanosis, and his rate of respiration was 82. A hot bath was given immediately, and he was put to bed with hot bottles around him, and with a turpentine stupe upon his chest. He rapidly recovered, smiled, and seemed quite comfortable.

May the 25th.—His temperature was 99·2° F., pulse rate 124, and respiration 30 to the minute. He had a good colour, but his cough was very troublesome, and

respiration was accompanied by an indrawing of the whole subcostal region during respiration, and much wheezing during expiration. There was no movement of the alæ nasi. Slight dulness and diminished breath-sounds were present over the right lung posteriorly, suggesting collapse of the lung, and a turpentine stupe was accordingly put over that region. Rhonchi were present in large numbers over both lungs in front and behind. The abdomen was very rigid and much distended. The edge of the liver was not felt.

May the 26th.—After a bout of crying he apparently fell asleep, but was noticed to be sweating profusely, and had marked cyanosis and distress. Artificial respiration quickly improved the colour, and the child breathed easily. Two similar attacks occurred during the day, which were relieved when the child passed flatus.

May the 27th.—At 11.30 p.m., with no previous exertion, he was noticed to have become cyanosed, and to be suffering from great inspiratory difficulty and expiratory stridor. The lower interspaces were indrawn upon the left side, but not upon the right, and air hunger was extreme. He rallied, but died in a second attack at 1.45 a.m. on the 28th.

Post mortem.—He was a well-developed child, with firm, well-formed muscles. The fontanelle was nearly closed. The liver nearly reached the symphysis pubis, the presenting part, on opening the abdomen, being the upper and anterior surfaces. The gall-bladder was not seen. The stomach occupied the remaining part below. The lower part of the pleural cavity upon the right side was seen to contain nothing but intestine, the superficial coils of which were distended, and those behind collapsed. The heart and pericardium were pushed over entirely to the left side. The upper lobe of the right lung, pale and firm, was the only part of the two lungs to be seen. The left lung appeared to be healthy. The middle and lower lobes of the right lung were collapsed and slate-grey in colour and had probably never been expanded.

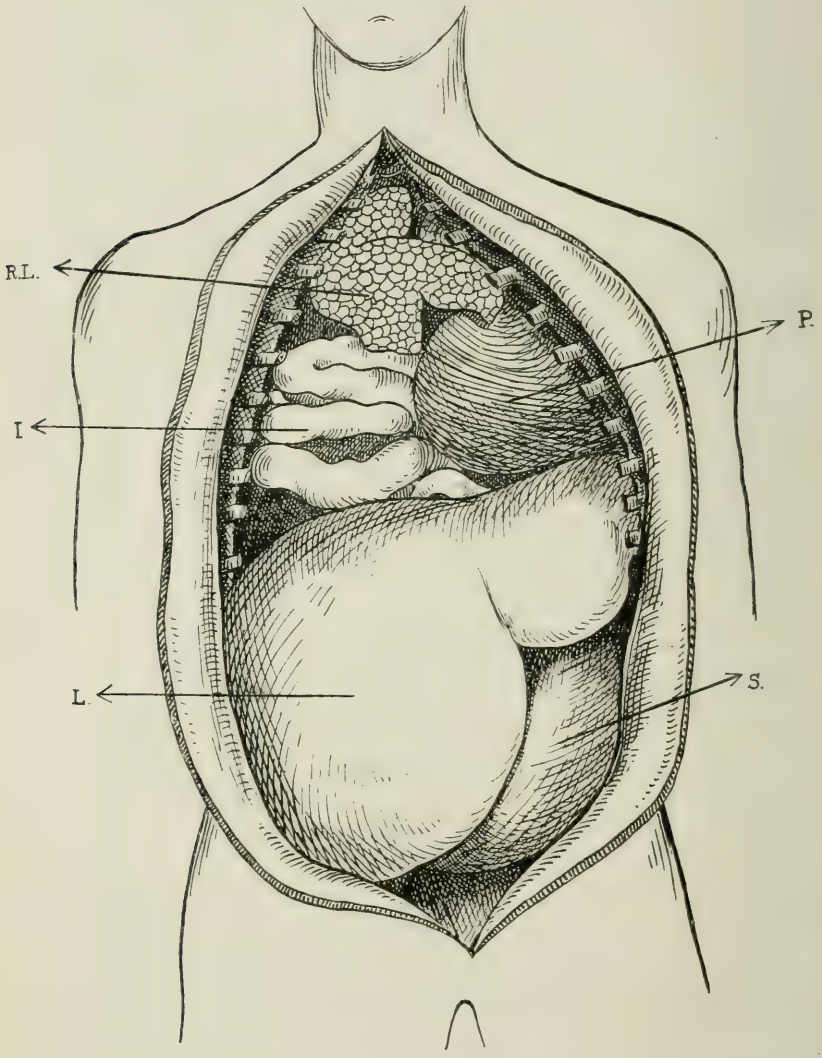

FIG. 9.—CASE 2.—R.L. Right lung. I. Intestines. L. Liver. P. Pericardium. S. Stomach.

The heart was normal, but the right pulmonary artery and veins were very small. The opening in the diaphragm was situated about two inches from the middle line and did not involve the right crus. It lay opposite the attachments of the diaphragm to the tenth and eleventh ribs, and admitted three fingers readily. It was circular in outline. There was no sac of any kind, but the parietal layer of the pleura and the peritoneum were in direct communication through the opening. The abdominal viscera present in the right pleural cavity consisted of the third part of the duodenum with the tail of the pancreas, the jejunum, ileum, cæcum, and ascending colon. The duodenal end was firmly adherent by its mesentery to the posterior part of the ring. The whole of the mesentery of the small and large intestine was abnormally long, and the hepatic flexure of the colon was attached to the anterior part of the ring by a firm band. There was no strangulation. The spleen and kidneys were not displaced, and the liver was not deformed, but was merely rotated upon a horizontal transverse axis.

It would seem that the diagnosis of such a condition should be comparatively easy. One would expect to find the marked displacement of the liver, and to have a tympanitic note and diminution of the breath-sound over the lower part of the right side of the thorax. Unfortunately neither child was in a condition suitable for thorough examination. The first was moribund on admission and evidently had only a few hours to live, whilst the second child cried whenever he was examined and soon got acutely distressed. The loud rhonchi present prevented any exact auscultation. In neither case was the diagnosis of diaphragmatic hernia thought of. In Case 2 the abdomen was rigid and distended, and the edge of the liver could not therefore be felt readily. The physical signs in the thorax are interesting to note. The collapsed lung at the back gave an area of diminished resonance with diminished breath-sounds, and the child's respiratory distress was thought to be largely due to this collapse. The note in

front was not markedly tympanitic, as might have been expected. This is probably due to the fact that as the child was examined in the intervals of comparative comfort, there must have been very little flatus present in the intestines. This is shown by the fact that the child's distress was so frequently noted to have been relieved by the passage of flatus. The adventitious sounds heard all over the chest obscured the true breath-sounds. The child had been sent in by the doctor, according to the mother's statement, for an operation for fluid in the right side of the chest.

A case of diaphragmatic hernia upon the left side with the stomach and part of the intestines in the thoracic cavity was recently operated upon in one of the children's hospitals for a supposed left empyema, although repeated exploration had failed to confirm the diagnosis.

Hillier showed a specimen, before the Pathological Society in 1861, of a diaphragmatic hernia upon the right side with the colon within the thoracic cavity. The child had had attacks of dyspnœa since she was two weeks old and died at seven months with no intestinal symptoms. In this case the note was dull at the lower part of the thorax on the right side and tympanitic above. The absence of any intestinal symptoms in all these cases is rather striking.

An indication of the cause of such hernia may be obtained from a consideration of the development of the diaphragm. Five elements—one median, two ventro-lateral, and two dorso-lateral—enter into the formation of the diaphragm. The median and ventro-lateral are first developed and together form the septum transversum. At a later period the two dorso-lateral elements, which are only present in mammals, are formed and unite with the septum transversum to form the "primitive pleuro-peritoneal septum," and completely shut off the pleural from the peritoneal cavity. It is easy, therefore, to understand why congenital diaphragmatic herniæ should almost invariably occur at the posterior part of the diaphragm.

After the second or third month of intra-uterine life the diaphragm is completed by the growth of muscular fibre springing, firstly, from around the œsophagus and, secondly, from the chest wall at the periphery of the diaphragm.

Both of these herniæ must be included among those classed by M. Nau as "embryonic diaphragmatic herniæ" —*i.e.* herniæ formed before the complete closure of the "pleuro-peritoneal septum," and those which have not therefore acquired a sac. He divides this class into complete, where the whole of the dorso-lateral element, or pillar of Uskow is deficient; incomplete where it is partially absent, and complex where the opposite one is also deficient. These cases will, therefore, come under the division of "incomplete embryonic diaphragmatic hernia."

M. Paillard in his Paris thesis of November, 1903, has put together statistics obtained by Bowditch, Lacher, Kohn, and Boursier, and has classified 130 cases which he has himself collected. In a total of 481 he has found that 93, or practically 20 per cent., have occurred upon the right side. Amongst his own 130 cases he describes 74 complete embryonic diaphragmatic herniæ, of which 15 occurred upon the right side, and in 14 of these the liver, much deformed, was partially within the pleural cavity. He has found only 16 cases of incomplete embryonic diaphragmatic hernia, and of these only 2 occurred upon the right side. One of these contained the small intestine, and the other the small intestine, cæcum, and right kidney. Thus in his 130 cases there were none similar to these two which presented such a remarkable resemblance to each other. It is a strange thing that rare conditions so frequently occur in pairs in a hospital practice.

I have to thank Dr. Guthrie and Dr. Sutherland for permission to publish these cases, and Dr. Emery for the notes of the post mortem in Case 1.

The drawing of Case 1 was taken from a photograph, that of Case 2 from a sketch made at the post mortem.

(*Read October the 21st*, 1904.)

16. CONGENITAL ATRESIA OF THE TRICUSPID VALVE.

By BERTRAM M. H. ROGERS, M.D.,

AND

J. M. FORTESCUE-BRICKDALE, M.D.

The child from whom this specimen was taken was admitted into the Children's Hospital, Bristol, on October the 3rd, 1904, under Dr. Rogers, and died there in a few hours. The mother stated that the child was 2 years old, and had appeared to be healthy till about 6 weeks old, when it had an attack of cyanosis. These attacks recurred once or twice a month (perhaps oftener) and lasted from a few hours to a whole day. It had never attempted to walk and had not yet been weaned. On admission the child was intensely cyanotic and gasping for breath. The fingers and toes were clubbed and the lips and extremities deep purple in colour. The temperature was 96·4° F., pulse 104, respirations 48. Physical examination showed that the lungs were full of loud rhonchi. The heart's apex beat was in the fifth space in the nipple line. No increase in the area of cardiac dulness could be detected, and no cardiac bruits were heard.

A post-mortem examination was made fourteen hours after death. The child was very small for its age and weighed only 11 lbs. 14 oz., but it appeared fairly well nourished. There was extreme congestion of all the veins and considerable bronchitis. The heart was the only other organ showing anything abnormal. It weighed 3½ oz., and its walls, especially the right auricle and left ventricle, were thickened. The auricles and left ventricle were also dilated, but the right ventricle could be seen before section to be very small. On opening the heart the auricles were found to communicate through the foramen ovale, the aperture being about 5 × 3 mm. and roughly oval in shape. The ventricles also communicated through a small opening

just below the aortic valves about 2 mm. in diameter. The right ventricle was very small, and was separated completely from the dilated right auricle by a smooth membranous septum into which the chordæ tendiniæ were inserted. There was no sign of endocarditis. The remaining orifices were normal, and the great vessels occupied their ordinary positions. The ductus arteriosus was impervious. The blood entering the right auricle, therefore, passed directly into the left auricle and thence into the left ventricle, whence some was pumped into the aorta and some through the small opening into the right ventricle and so into the pulmonary artery.

This condition is rare. Holt out of 242 cases of congenital malformation of the heart collected from the literature found atresia or stenosis of the tricuspid valves in only three, so that at most complete atresia could only have been observed in two. In most of the cases we have found described there was much more deficiency in the interventricular septum, and in some abnormalities of the great vessels are also described. Sievking's case, described in the 'Pathological Transactions' in 1853, and now in the Museum of St. Mary's Hospital, resembled this case somewhat, but there was much freer communication between the ventricles. This child had practically no cyanosis, and there was a loud systolic bruit apparently heard all over the præcordia. The child suffered from symptoms of collapse, with much dyspnœa, and died at the age of 9 weeks. A comparison between these two cases is interesting; in both the course of the blood-stream through the heart was identical, only in our case the amount of blood reaching the pulmonary artery was presumably less. The cyanosis was well marked in the present case, whereas in Sievking's it was practically absent. This, we think, is explained by the very small volume of blood which could pass into the pulmonary artery and consequently the very deficient aeration. The cyanosis and clubbing in this case are also, no doubt, accounted for by the obstruction at the interauricular septum, as there was practically no obstruc-

tion to the exit of blood from the left ventricle, the aorta being normal. The amount of blood, too, reaching the left auricle cannot have been in excess of normal, as it practically all came from the right side of the heart through a small opening, the pulmonary vein contributing but little. The hypertrophy and dilatation was, no doubt, started in the right auricle and affected the left side of the heart chiefly through the systemic circulation, but in addition the rapid and violent action of the heart set up by any exertion in order to drive more blood through the opening in the interventricular septum probably contributed to the hypertrophy of the left ventricle.

The duration of the child's life under these conditions seems remarkably lengthy (two years), but Peacock mentions a case said to be anatomically similar to Sievking's in which the patient survived to the age of 9 years.

(*Exhibited November the 18th*, 1904.)

17. A SPECIMEN OF CONGENITAL MORBUS CORDIS.

By GEORGE CARPENTER, M.D.

The specimen was removed from a female, aged 2½ years. She was an only child. The birth, which was premature, was said to be at 6 months, and she was born blue and continued to be cyanosed; but 9 days before she came to hospital the cyanosis increased. At that time she had an attack of dyspnœa followed by a convulsion. When 12 months old she had a convulsion for the first time.

When she came under observation, on May the 6th, 1904, her lips and finger-tips were very cyanosed, and she presented marked clubbing of the fingers and toes. There was no history of congenital syphilis, and the child was free from congenital abnormalities other than the heart lesion.

The cardiac impulse was most marked in the epigastric notch; there was an impulse in the fifth left interspace 1½ inches internal to the nipple line and an impulse also 2 inches to the right of the sternum.

The cardiac area of dulness extended well to the right side of the sternum for a couple of inches, and a skiagram verified the diagnosis.

Sounds.—A loud systolic bruit was audible all over the cardiac area, of greatest intensity over the xyphoid cartilage. The bruit was not audible in the great vessels of the neck; it was sometimes audible in the back, but not at others. Urine free from albumin.

Blood-count.—Red cells, 7,880,000 per cmm. White cells, 9750 per cmm. Hæmoglobin, 122 per cent. There were no other abnormalities.

On May the 8th she had a short attack of dyspnœa, which did not end in convulsions. On May the 17th she developed measles, and on May the 23rd she died.

Post-mortem.—Both lungs were œdematous and showed patches of broncho-pneumonia. The spleen was hard. The liver was cloudy and in parts fatty. Peyer's patches and solitary follicles were swollen and markedly injected. The cortical vessels of the brain were enormously distended, its substance very vascular and pink in colour.

Heart.—A gap between the annulus ovalis and the auricular septum through which the little finger can be passed. At the upper and lower part of the membrane there are also two small holes. The pulmonary orifice is narrowed, the vessels small, and the walls of the right ventricle are much hypertrophied. There is a perforated ventricular septum, and the aorta, which is enlarged, is superimposed over both chambers.

There are several points of interest about the case, firstly, the polycythæmia and the largely increased percentage of hæmoglobin—a condition which I have several times observed in congenital morbus cordis with cyanosis. Next is the early advent of cyanosis; it so often happens that cyanosis is absent in the first few months or even years of

cardiac malformations. Lastly, the absence of a systolic bruit in the great vessels of the neck. When there is a perforate ventricular septum, it more often happens that the bruit is plainly audible in these vessels, and when a bruit is heard there during life it is a strong point in favour of a diagnosis of perforate ventricular septum.

(*Exhibited November the 18th,* 1904).

18. A SPECIMEN OF CONGENITAL MORBUS CORDIS.

By SYDNEY CURL, M.D.

The specimen was removed from a child, aged 3 months. As regards the clinical history of the case very little is known. There was no lividity or any other sign leading one to suspect congenital malformation of the heart and no sign of congenital syphilis was present. The patient suffered also from a hare-lip and cleft palate, and died from some acute infection (the nature of which was doubtful) before operative interference could be undertaken for the latter deformities.

The heart shows the following abnormalities:

(1) Deficiency of the ventricular septum at the upper part.

(2) Incomplete formation of the auricular septum, the two auricles forming one chamber.

(3) Complete atresia of the pulmonary artery soon after its origin from the ventricle.

(4) A large aorta springing from both ventricles, but chiefly from the right.

The specimen is of interest as it illustrates very well the rare condition of atresia of the pulmonary artery. This condition as far as I am aware is exceedingly rare, although moderate degrees of stenosis are comparatively common.

For the clinical abstract of the case I am indebted to

Dr. Powell, house-surgeon at the North-Eastern Children's Hospital, and for permission to publish the case my thanks are due to Mr. E. Stabb, under whose care the child was admitted.

(*Exhibited November the 18th,* 1904.)

Discussion.

Dr. Edmund Cautley said the cases shown (Nos. 16, 17, and 18) were of extreme interest from the developmental point of view, and he would have liked to have heard from the exhibitors their opinions on the causation of these defects. Complete atresia of the tricuspid valve was certainly extremely rare, and also so was the kind of case shown by Dr. Curl. All three of the cases showed defects which were due to persistence of conditions existing during the first two months of life. One knew the septum ventriculorum was completed by the end of the seventh or eighth week. Congenital heart disease was often ascribed to rheumatic fever, but it seemed impossible this could be the explanation of these cases. It was more probable one had to do with some congenital defect of development. He asked each exhibitor whether there had been a history or evidence of syphilis in any of the cases. He believed syphilis to be the great cause of congenital heart disease. Frequently there were other developmental defects present. He confirmed the statement of Dr. Carpenter as to the excessive number of red cells and of hæmoglobin in cases of congenital heart disease. This character of the blood was often found, but it was not obvious why.

Dr. Fortescue-Brickdale (Bristol), in reply, said that in his case there was no evidence of syphilis, as far as he could make out. There was no syphilitic history in the family.

Dr. George Carpenter, in reply, said he was in favour of the view expressed by Dr. Cautley as to congenital syphilis being responsible, but he thought only for a certain number of cases of malformations of the heart. He had encountered cases in which that evidence was undoubted, but errors of development, intra-uterine rheumatism, and other blood infections were very frequent causes.

Dr. Sydney Curl, in reply, said there was no evidence of syphilis in his case, but there were other congenital abnormalities.

19. A CASE OF JUVENILE GENERAL PARALYSIS.

By JAMES TAYLOR, M.D.

The patient who is the subject of this paper is a boy, aged 11½ years, who was referred to me by Mr. Treacher Collins, whose advice he had sought at Moorfields Eye Hospital on account of failing vision. Mr. Collins has kindly allowed me to use the case, and I am indebted to his notes for many of the particulars.

The boy is a fifth child in a family of nine, all of whom are said to be healthy. The father and mother are also in good health, and there have been no miscarriages. The child was born at full-time, and at the age of 12 months is said to have suffered from abscesses in the scalp and also in the groin. He was very backward and did not walk until he was 4. At 5 he went to school and is described as having been dull and inattentive. At 6 he apparently first showed distinct mental peculiarities. He developed a faculty for romancing, told many extraordinary stories apparently without any object except that of exciting wonder, and was occasionally quarrelsome and very violent. He was very fond of going to school or church, and had reached the third standard. In the past year he had complained of constant headaches, and his violence and quarrelsomeness had become worse. He also had had some difficulty in walking, but this had not been very great. He was fond of talking of himself, was persuaded he was a great singer, and was in the habit of collecting other children to listen to him. Three months ago his sight began to fail, and it was on this account that he was brought to Moorfields.

He is a fairly well developed boy, without any definite stigmata of congenital syphilis, but his facial appearance is distinctly suggestive of such a condition. He is pale, rather quiet, unusually pleased with himself, and not markedly unintelligent. He walks rather feebly, and there is a very

slight degree of ataxy. His knee-jerks are active; the plantar reflexes are extensor in type. His muscles are fairly developed, and his heart and lungs are normal. There is no albuminuria. There is no interference with articulation; the tongue is protruded in the middle line and is not tremulous. There is a slight tremor of the facial muscles occasionally when he shows his teeth. The pupils are unequal, the right being the larger; there is no reaction to light in either, but there is a reaction during convergence. His vision is much impaired. With the right eye he can only distinguish light from darkness; with the left he counts fingers at six feet. There is well-marked optic atrophy in each disc.

There seems no reason to doubt that this is a case of juvenile general paralysis. The mental peculiarities and the physical signs enumerated make the diagnosis almost certain. As regards the mental condition cases of juvenile general paralysis fall into two distinct groups. In one the children have always been backward, and a condition of progressive dementia is, so to speak, engrafted on a mental condition which has never been normal. In the other class the children are unusually sharp and intelligent, winning numerous prizes, and are indeed of the same intellectual type as the adult general paralytic. In this type the onset of symptoms, at or about puberty, is usually determined by an acute illness or accident, but it may come on gradually and apparently spontaneously. Our patient belongs to the former class. In regard to his physical signs he is not of the usual type, with tremor fits and paraplegic weakness. Slight tremor and slight weakness of the lower limbs are present, it is true, but the most striking condition is the optic atrophy; and just as in the adult disease and in tabes dorsalis also, when this is the first symptom, it may remain for a long time unassociated with any other.

The relation of syphilis to juvenile general paralysis is a very close one. All evidence seems to show that syphilis is an almost constant precursor of the condition. The

majority of cases of the disease actually carry undoubted stigmata of congenital syphilis—scarring about the mouth, Hutchinson's teeth, choroidal atrophy, or interstitial keratitis. This patient has none of these, and has a family history free from suggestiveness. Yet the meagre physical development, the facies, which, as I have said, is suggestive, and the Argyll Robertson pupils, if not conclusive, are sufficient to raise suspicions. In 13 of Mott's 22 cases there were definite stigmata of syphilis, and it could not be excluded in any of the others. Thiery also found it in 64 per cent., a proportion which, he says, is too great to permit of syphilis being regarded as occupying merely a secondary *rôle* in the production of the disease.

The prognosis in such a case as this is necessarily bad. Vision is likely to be completely lost, and the mental and physical weakness is almost certain to increase. No drugs seem capable of even modifying the course of the disease.

(*Exhibited November the 18th,* 1904).

Discussion.

Dr. F. Parkes Weber remarked that in 1898 he saw a boy, aged 19 years, who had the symptoms of general paralysis of the insane. He had been perfectly normal until he reached 17½ years of age, and at that age began to have tremors, and gradually lost ground physically and mentally. When Dr. Weber saw the patient, one and a half years after the commencement of the disease, the tremors formed a striking feature of the case and were much more intense than the ordinary tremors of general paralysis; they affected all the limbs and were somewhat diminished by voluntary movements. When Dr. Weber saw the patient again in January, 1900, he was confined to bed, helpless and apathetic, and would pass all his motions and urine under him. The lower extremities were rigid, and there was decided ankle clonus. There was tremor in the tongue and hands on movement. Death occurred in June, 1900. Dr. Weber asked whether the tremors had any special significance, and whether excessive tremors justified the supposition that in such a case the child suffered from an active inflammatory process (possibly of a genuine syphilitic nature) affecting the cerebral cortex. In favour of that supposition was the rigidity of the legs which occurred towards the end of the disease.*

* In fact, probably owing to great involvement of the Rolandic areas, the symptoms of a progressive cerebral diplegia (with spastic para-

Dr. James Taylor, in reply, said he thought cases of general paralysis were divisible into two classes—(1) that in which the child was never quite normal; (2) that to which Dr. Parkes Weber referred, in which the child was not only normal, but unusually intelligent and well, up to a certain stage, after which degeneration set in and progressed rapidly. With regard to tremor, he did not think that was by any means a constant early symptom of general paralysis. In the adult, optic atrophy, and Argyll Robertson pupils might be the only sign of some such change for a long time, and then speech became defective, and tremor might be only a very late symptom. And tremor was not constant in children the subjects of the disease. It was comparable to tabes dorsalis in which, although ataxy was one of the common symptoms, there were many cases of tabes dorsalis in which that symptom was absent.

Dr. Parkes Weber said his question was as to whether excessive tremor indicated the presence, not merely of the changes of general paralysis, but of some more active inflammatory process in the cerebral cortex, possibly syphilitic.

Dr. James Taylor, in further reply, thought it was scarcely necessary to assume the presence of some special process to account for a particular degree of tremor; its extent seemed only to depend on the intensity of the general process.

20. TWO CASES OF OCCIPITAL CEPHALHÆMATOMA NEONATORUM.

By J. HOWELL EVANS, M.Ch.

Two cases of cephalhæmatoma neonatorum over the occipital bone were shown by Mr. J. Howell Evans, who subsequently gave a lantern demonstration on the subject. Mr. Evans considered these blood effusions were due to a reactionary hæmorrhage from a small artery which had been injured in the moulding of the infant's head during its passage through the birth-canal.

plegia) accompanied those of general paralysis in this case. If the same pathological process ever were, as possibly it might be, limited to the Rolandic areas, the resulting clinical features would be, not those of general paralysis, but those of a progressive cerebral spastic diplegia.

Fœtal skulls and many lantern slides of the skulls of various animals were shown to illustrate Mr. Evans's original views. The author advocated the surgical treatment of cephalhæmatoma neonatorum, and drew attention to his former observations on this subject.*

(*Exhibited November the 18th*, 1904.)

Discussion.

The Chairman (Mr. Robert Jones) (Liverpool) congratulated Mr. Evans on his interesting demonstration. He had had very little personal experience of cephalhæmatoma neonatorum, but six or seven years ago he was called to see a baby suffering from that affection and it was decided against operative interference. In the course of time the tumour quite disappeared and there was little or no trace of thickening. In the Out-Patient Department of the Stanley Hospital he remembered seeing a case of suppurating scalp resulting from an attempt to drain a cephalhæmatoma. The child was for a considerable time in a critical state, which showed how important it was that if the operation were performed it should be performed with the utmost caution. He suggested that if an operation were to be performed it would be almost better to wait and see what Nature would do first and if the thickening remained that might be treated by exsection. Newly-born babies were not very desirable subjects for operative interference.

Dr. C. W. Chapman said that years ago when he was seeing such cases the condition got absolutely well after being left alone. He would not recommend anything to be done in the way of operation were such cases to come before him again.

Mr. Howell Evans, in reply, said leaving those tumours alone was the advice which had generally been given and adhered to, but he could not find throughout the whole of the writings on the subject any person who had viewed the condition in the light of reactionary hæmorrhage. If it was right to treat surgically a reactionary hæmorrhage in the leg, why not operate for a similar condition on the delicate skull of a child? By prosecuting his studies further he believed that in time he would be able to prove that nearly all the spastic congenital hemiplegias of which neurologists wrote were due to neo-natal traumatisms associated with those very vessels which were passing from without the skull to its interior.

* "The Relation of Certain Extra- and Intra-cranial Hæmorrhages in the Newborn," the 'Reports,' vol. iv, pp. 100–104.

21. THREE CASES OF UNILATERAL CONGENITAL DISLOCATION OF THE HIP.

By DOUGLAS DREW, B.S.

Mr. Drew brought the cases forward with the idea of obtaining expressions of opinion as to the treatment. He thought in one case that no treatment, either operative or manipulative, was needed, as the child walked extremely well with the aid of a slightly raised boot. In the other case, in which the deformity was more pronounced and walking more difficult, Mr. Drew thought that operative measures would be needed, and expressed the opinion that he should choose the open operation in preference to the manipulative.

(*Exhibited November the* 18*th*, 1904.)

Discussion.

Mr. Laurie McGavin said his personal experience of that operation had not been great, but he had seen a large number of cases done from time to time; five cases which were operated upon four or five years ago came to his recollection which had some bearing on the point raised. They were all operations performed by recognised heads of the profession in London, were unilateral cases, and done by the open operation and within three weeks of each other. They varied very much in the degree of displacement, and three of them at least were cases which certainly were not very markedly affected; in fact, one boy when waiting in the front surgery with his father suddenly became terrified, ran off, and was only with difficulty caught. They were all submitted to the open operation, and three of them died as the direct result, one from shock and two from septic sequelæ. One got septicæmia and died in four days, and one was pyæmic. Of the two which recovered, one relapsed completely to its original condition, and indeed was rather worse than before, and the last had ankylosis of the hip. Whether that ankylosis would be of benefit in after-life remained to be seen. When one considered the possibility of such disastrous results accruing from such operations on young children with only a slight defect of locomotion, it seemed a somewhat unjustifiable operation, and unless one could guarantee a moderately favourable termination one should be careful about submitting children to it. It was

very difficult to keep the parts clean in young children, and septic consequences were very apt to occur, with the very probable death of the child. The chance of retaining the head of the bone in the acetabulum was very slight. When a wire or screw had been put through bone, and the fragments were put into position, there was a tendency (often seen in the tibia) to erosion of the hole through which the wire was passed, resulting in the latter becoming loose before firm callus was formed. However unsatisfactory Lorenz's operation had been, one should be careful how one submitted young children to the open operation when the results were so doubtful.

The Chairman (**Mr. Robert Jones**) (Liverpool) said the subject of the treatment of congenital hips was so large that a general discussion of it could not be entered upon then and there. He had seen bad results after manipulation and bad results after operation. Catastrophes had been related by the best surgeons who operated by the open method, and fractures of the neck of the femur and worse troubles sometimes occurred after attempted reductions by manipulation. He thought all would agree that if the little child brought forward by Mr. Drew walked as well fifteen or twenty years hence as he did now, no one would ever think of adopting any treatment either manipulative or operative. Unfortunately, however, that was not the history of such cases. He constantly saw patients with very limited abduction, with pain in the joints, who came for relief of symptoms when between 15 and 30 years old; and at the present moment he had been obliged to operate upon a young woman aged 30 years by removing both femoral heads because of the degenerative and inflammatory changes that had taken place in consequence of their having been congenitally dislocated. Surgeons had not lived sufficiently long to know even yet what would be the after-results in cases reduced by manipulation by the Lorenz method, nor can positive final results be quoted with regard to the open method. It should be remembered that Professor Hoffa, who was one of the pioneers and did yeoman service by the open method, had now practically discarded it in the case of children and practised largely a special manipulative process. Lorenz himself, after operating on some hundreds of cases, had also found refuge in his so-called "bloodless" method. Why was this? Because in spite of every care the results were not sufficiently good to justify the risks. This was an extremely important fact and must bear considerably more weight than the reports of isolated operations which were every now and again published. One should not hurriedly come to any conclusions, but rather patiently wait until sufficient time had elapsed for results to be verified. Personally he was very much in favour of the Lorenz method. He had performed it over 80 times, and

at the Oxford Meeting of the British Medical Association had shown radiographs exhibiting results in 35 cases. He had had some bad results where stiff joints resulted and rotated feet, and some cases where the neck of the femur had been broken; but he recognised they were due to his having chosen the wrong class of case for the operation, to his not having thoroughly mastered the niceties of manipulation, and to his not having been sufficiently energetic in the matter of early massage. The two cases operated upon by Lorenz in Liverpool he had examined quite recently: they were both absolutely all right and exhibited perfectly free movement. In a third case (a double one) anterior reposition took place on one side and a perfect reduction on the other. He thought that often a personal feeling was thrown into this controversy, which did not tend either to careful observation or to accuracy of statement. If because of a hopeless acetabulum he decided to operate he would prefer first of all reducing the hip by manipulation, keeping it in position for two or three weeks, and then cutting down anteriorly and releasing any fibrous obstruction that prevented accurate adaptation. This, he thought, would be better than harsh manipulation in presence of an open incision.

22. A CASE OF OLD-STANDING DISLOCATION OF THE HEAD OF THE RADIUS.

By DOUGLAS DREW, B.S.

The dislocation was forwards and was reduced by operation, but, in spite of the greatest care, the condition recurred as soon as movement was permitted. Inasmuch as the function of the arm was very little impaired and the deformity was trifling, Mr. Drew advocated no further interference.

(*Exhibited November the 18th*, 1904.)

Discussion.

The Chairman (Mr. Robert Jones) (Liverpool) said the case particularly interested him because he had seen a great number of cases of dislocations forward of the radius, both ancient and modern, and he could confirm what Mr. Drew said as to the difficulty in keeping the head in position. The

best way to reduce the deformity was to supinate the arm, put pressure on the head of the radius, and then fix the arm in the acutely flexed position : a recurrence might even then sometimes result. The interesting practical point, however, was that if recurrence did take place it made very little difference as regards the utility of the arm. In nearly all cases where no complication existed the range of movement was thoroughly good whether reduction had been effected or not. He certainly felt that the condition was not worth while running the risk that an operation entailed. He remembered a catastrophe as far as movement was concerned after wiring in such an accident. It is interesting to note that in some old cases one can quite easily by putting pressure on the head of the radius reduce the deformity which of course immediately recurs.

23. A CASE OF BELL'S PALSY WITH HEMIPLEGIC ONSET.

By J. PORTER PARKINSON, M.D.

This boy, aged 4 years, was put to bed three months ago apparently well, and slept as usual. On being taken out of bed in the morning and placed on the floor he screamed and fell down. On being seen by a doctor he was found to be paralysed in the right arm and leg and the left side of the face. There was also general hyperæsthesia. The paralysis of the arm and leg passed away in a week, and he was brought to see me a fortnight ago for the paralysis of the face. He is a healthy-looking boy, with no evidence of any disease beyond the paralysis of the face, which affects both the upper and the lower parts equally. There is no affection of any other cranial nerve, and no loss of power or wasting in the arms or legs, but the right knee-jerk is more active than the left. There is no ankle clonus on either side and the plantar reflexes are flexor. He walks perfectly. There is no evidence of disease of the heart or any other organ.

There can be no doubt that the lesion is in the left side

of the pons, and I would suggest that this may be an example of encephalitis inferior, pathologically similar to anterior poliomyelitis in which the stress of the disease has fallen upon the facial nucleus, while the adjacent pyramidal tract was affected by shock. The acute onset of the disease in a child who appeared in perfect health, without symptoms of serious and extensive disease of the pons, and the preliminary hyperæsthesia are in harmony with this view, but I have not given this heading to the case as I hope to hear the opinion of other members of the Society.

(*Exhibited November the* 18*th*, 1904.)

Discussion.

Dr. Edmund Cautley said he thought Dr. Porter Parkinson's diagnosis depended mainly on the history, and it was always difficult to accept histories of that nature, especially in such an unusual type of case. Personally he mistrusted histories. He thought the child had got a mild attack of hemiplegia on the right side, and that during the course of it he developed a typical Bell's paralysis. If there were a lesion higher up in the nerve, or in the nucleus, he did not think there would be such a good nutrition of the face as could be seen at present.

Dr. Porter Parkinson, in reply, agreed with Dr. Cautley as to the danger of accepting histories, but he thought there was danger in accepting the view that the symptoms were due to hæmorrhage. For instance, the paralysis of the arm and leg passed off in a week and had left no remains, and there was no increase of reflexes on that side, which was usually the case after such a condition. With regard to the position of the lesion in the nucleus, he believed it to be there because the facial paralysis came on at the same time as the arm and leg paralysis. He had examined the ear and other places in which one might suspect disease, but there was no sign of any such disease, and the child was absolutely healthy except for the paralysis on the left side of the face. He attributed the crossed hemiplegia to the shock of the lesion in the facial nerve. It evidently was a very temporary affair. There was no history of any illness pointing to a serious lesion in the pons.

24. A CASE OF ATAXY OF THE CEREBELLAR TYPE.

By J. PORTER PARKINSON, M.D.

I have made use of the above heading as pointing out the most prominent symptom present and not as suggesting that the main disease is in the cerebellum. This appears to me to be a case of degeneration occurring in a patient who was most probably affected by inherited syphilis, in whom the symptoms point especially to disease in the neighbourhood of the corpora quadrigemina. I saw the girl, whose age is 11 years, at the North-Eastern Hospital for Children. Her recent personal history was good, having only suffered from whooping-cough and measles when under 3 years of age. She is, however, subject to "bilious attacks." The mother has three other children, all healthy, and has had no miscarriages. The father and mother are both living and in good health. When 7 years of age this child was noticed to totter and stagger when walking and this has gradually got worse up to the present time. For the last three or four months the sight has been failing, and at times she was unable to distinguish objects when passing from a well-lighted to a darker room. She is backward at her lessons and is distinctly mentally dull, and rather lachrymose. She has not suffered from headaches.

On admission into the hospital she was proved to be healthy-looking but thin. She walks with a very ataxic gait, the feet being planted wide apart and coming down on the heel in a very uncertain fashion. She occasionally falls if unsupported, but not in any special direction. She can stand well with the eyes closed. The patella reflexes are increased, but there is no ankle clonus. The plantar reflexes are distinctly extensor in type. There is much inco-ordination of the arms and hands and the supinator and triceps reflexes are increased. There is no wasting or rigidity of muscles and the cutaneous sensation and special senses are normal. Mr. Sydney Stephenson kindly ex-

amined the eyes and found their upward and lateral movements to be distinctly limited, especially the former. Vision is impaired, being under atropine R.V. $\frac{1}{24}$ ij; L.V. $\frac{1}{24}$ ij. Jaeger type No. 14. Ophthalmoscopically the retinal vessels are of good size in both eyes and the coats show no signs of degeneration. The fundus of both eyes is universally mottled with indistinct blackish patches of choroido-retinitis such as is not infrequently found in cases of congenital syphilis. In the left eye the disk is practically encircled by an area of choroidal change. There is no optic neuritis or atrophy.

These symptoms seem to point to some affection in the region of the corpora quadrigemina which will explain the ataxy, the oculomotor paralysis, and the increased reflexes, but besides this there is doubtless a more diffuse lesion producing the mental symptoms.

The slow onset and progressive character of the symptoms points to degeneration, while the retinal changes suggest a syphilitic origin, of which, however, there is no other evidence either direct or indirect. She is at present being treated by mercury.

This case differs in many respects from congenital cerebellar ataxy, notably in the presence of oculomotor paralysis, and most nearly resembles a case described by Dr. Spiller, of Pennsylvania in 1896 ('Brain').

(*Exhibited November the 18th*, 1904.)

Discussion.

Dr. C. O. Hawthorne agreed with the suggestions of Dr. Parkinson. With regard to the fundus condition, if one was justified in forming a conclusion from a single observation, he would say it was strongly suggestive of hereditary syphilis.

25. A CASE OF GRAVES'S DISEASE IN A BOY TWELVE YEARS OF AGE.

By SYDNEY STEPHENSON, C.M.

Two instances of Graves's disease in children have been reported to the Society for the Study of Disease in Children. The first, shown in 1903 by Mr. Arnold Lawson,* occurred in a well-nourished girl, aged 12 years, who presented exophthalmos, enlargement of the thyroid—in a word, to quote the author, "the typical characteristics of a patient in an advanced stage of Graves's disease, excepting that she has never lost flesh and shows no trace of anæmia." The second case was shown at the Provincial Meeting at Bristol in 1904† by Dr. E. Cecil Williams. The patient, a girl, aged 12 years, was breathless on exertion, there was some exophthalmos, the pulse was 160 to the minute, and slight muscular tremor existed. There was no enlargement of the thyroid, neither was Graefe's sign elicited.

The case now shown makes, then, the third so far brought under the notice of the Society.

Arthur L—, aged 12 years, fell under my notice on May the 16th, 1904, on account of the condition of his upper eyelids, which were stated to have become swollen about a year before I saw him. The swelling had subsided, but had recurred. There was a history of a former attack of the same nature, affecting both eyes, and lasting one week. A diagnosis of angio-neurotic œdema was made. Four months later, however, the clinical picture had changed. The upper lids were still slightly puffy; but the thyroid was then found to be moderately enlarged; the pulse-rate was 120 to the minute; and there was a slight degree of exophthalmos. The visual field for white was contracted. On the other hand, the findings were negative as regards tremor, sleeplessness, von Graefe's sign, and Moebius's phenomenon.

I would ask members whose experience of these cases is

* 'Reports,' vol. iv (1904), p. 58.

† *Ibid*, p. 316.

greater than my own whether Graves's disease in children presents any peculiarities, particularly with regard to prognosis and treatment. The cases I have personally seen in people over 50 years of age have always appeared to me to be of an atypical kind. It is possible that such may be also the case at the other end of the age-scale.

(*Exhibited November the 18th,* 1904.)

Discussion.

Dr. G. A. Sutherland said he supposed comparatively few saw cases at the age of the present child, or even a few years later. The only case which he remembered seeing was that of a girl, aged 13 years, who suffered from severe attacks of chorea and from symptoms of Graves's disease afterwards. He had had her under observation seven years, and the symptoms had gradually subsided. There was remaining some exophthalmos and enlargement of the thyroid, but apart from that she was quite healthy, and indeed was a very stout robust girl.

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26. INJURY TO THE CORNEA FOLLOWING INSTRUMENTAL DELIVERY.

By SYDNEY STEPHENSON, C.M.

Paulina H—, aged 12 years, was brought to me at the Evelina Hospital, on November the 1st, 1904, on account of defective sight. R.V. $< \frac{5}{60}$; L.V. $\frac{5}{12}$. Atropine was prescribed, and a week later the girl's eyes were examined. The right cornea was then found to present two faint, grey, curvilinear lines, neither exceeding 0·5 mm. in width, running parallel with one another across the lower half of the cornea. The appearance of these curious marks was so strongly reminiscent of that in the cases described by Drs. Thomson and Buchanan as following instrumental delivery that I questioned the mother with regard to the details of the patient's birth. I then ascertained that the mother—a small woman—was 35 years of age when her first child, the

patient Paulina, was born. The labour was a long and difficult one, and was eventually terminated by forceps applied under chloroform by the two medical men in attendance. The baby, when delivered, was "as black as a coal," and was indeed believed at first to be dead. The right eye was noticed to be nearly shut, but the lids slowly assumed a more normal appearance under the influence of a domestic remedy in common use amongst the lower classes in London, namely, a tea-leaf poultice. There have been three living children and two miscarriages since Paulina was born. This history, together with the peculiar lines found on the patient's cornea, left no doubt in my mind that I was dealing with an instance of traumatic keratitis.

To complete the record, it should be added that the vision of the affected eye could be brought to $\frac{5}{12}$ with the following correction: — 1·25 D. sph., with — 1·50 D. cyl. axis 30°. Much irregular astigmatism was present.

(*Exhibited November the 18th*, 1904.)

Discussion.

Dr. C. W. Chapman asked whether any albumin was found in the urine, because there was a moderate accentuation of the second aortic sound and duplication of the second pulmonic sound. The heart did not appear to be enlarged.

Mr. Sydney Stephenson, in reply, regretted that the urine had not been examined.

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27. CASE OF PROGRESSIVE PALSY OF CEREBRAL ORIGIN.

By HARRY CAMPBELL, M.D.

Boy, aged 13 years. Mother has had five children. During the pregnancies of three of them there was uterine hæmorrhage. All these three were born prematurely, and they all had convulsions. The first of them was a seven months' child and died at 6 months; the second—the

patient—was an eight months' child; the third, also an eight months' child, died at 17 months, and seems to have been hemiplegic. The patient was born (delicate) at the eighth month. The mother had uterine hæmorrhage the whole time she carried him, and was in bed with hæmorrhage a week before the child's birth. He was bottle-fed, he suffered from convulsions, and no one thought he would live until he was 2 years old. He suffered every two to three months from attacks of vomiting and stupor, which lasted for some hours. He began to walk when 2 years, 5 months old. His head closed when he was 2 years, 4 months old. Thenceforward he steadily improved in health, and at the age of 7 was a strong, fine-looking boy. At this time he had a "kind of fit," which lasted about an hour He lost consciousness, opened his mouth, put out his tongue, which was black, and rolled his eyes about. The mother attributed this seizure to the great heat which prevailed at the time. He quite got over this attack, but a fortnight afterwards the mother noticed that the boy, who had hitherto walked normally, began to walk on the toes of his left foot. For weeks she thought it was only a habit, and was constantly telling the boy "to put his foot down and not walk like that." Some months after this he was taken to St. Bartholomew's Hospital, where his foot was put up in plaster, but without any improvement. Two years later, *i. e.* when he was 9 years old, he was admitted into the same hospital, under Mr. Walsham, who cut some tendons of the left foot, and this was followed for a few months by slight improvement in walking in the left foot. Three months afterwards the mother noticed that the left hand began to get drawn and the right leg to be affected like the left. When 10 years old, *i. e.* one year after his admission into St. Bartholomew's, he again saw Mr. Walsham, who said it was a hopeless case. It was now noticed that the muscles of the face were beginning to be affected, speech becoming difficult and indistinct, while the feet got so bad that he could hardly walk. When 11 years old, *i. e.* two years ago, he went into the

Orthopedic Hospital, Oxford Street, where the tendons of both feet were cut in three or four separate operations, but without any benefit. Soon after this the right hand began to get bad, and writing became difficult. (He went

Fig. 10.

to school at the age of 5 years and made good progress.) The mother says he has steadily been getting worse since the beginning of the paralysis, and that about every three months he has had a slight "faint," during which he turns white and giddy and vomits, but never quite loses consciousness.

Present condition.—He is a well-nourished, healthy-looking boy. The nails are bitten.

Face.—There is a generalised weakness of the facial muscles, especially on the right side. He finds great difficulty in elevating the upper lip, which permanently

FIG. 11.

covers the front upper teeth. The weakness of the muscles about the mouth causes an indistinctness of utterance.

There is no paralysis of the ocular muscles, the muscles of mastication, the soft palate, or of the pharynx.

Hands.—The paralysis seems to be almost entirely confined to the extensor longus digitorum. On the left side

there is marked secondary contraction of the large flexors of the fingers, especially of those belonging to the second, third, and fourth fingers. On this side also there is a tendency to athetosis.

Feet.— Both feet, but especially the right, are fixed in the position of equinus. The paralysis involves chiefly the extensors of the toes, the right hallux apparently escaping.

There is no wasting. The electrical reactions are normal ; the condition of the tendon-jerks and skin-reflexes calls for no comment.

Diagnosis.—The lesion in this case clearly involves the upper motor neurons, and the occurrence of the fits, especially of the one which heralded the onset of the paralysis, suggests that it is cortical. But for the progressive nature of the paralysis, the diagnosis would have been double hemiplegia from thrombosis. As it is the diagnosis would appear to rest between a progressive symmetrical thrombosis in the vascular areas of the motor cortex, or a progressive degeneration originating in the nerve-elements of the region in question.

(*Exhibited November the* 18*th,* 1904.)

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28. A SPECIMEN OF TUBERCULOUS ULCERATION OF THE INTESTINE AND TUBERCULOSIS OF THE RIGHT FALLOPIAN TUBE.

By SYDNEY CURL, M.D.

These specimens were removed from a girl, aged 2¼ years, who died of general miliary tuberculosis, and exhibit the following conditions.

Numerous ulcers are seen extending at irregular intervals from the upper part of the ileum to the cæcum. They are mostly small and rounded with indurated edges. A few are transverse in direction and oval in shape, and in the fresh

specimen tubercles were plainly seen on the peritoneum covering the ulcers. No ulceration was found in the colon.

The right Fallopian tube is much enlarged, and on section appears caseous throughout. The other tube is normal in appearance, and the uterus and ovaries are also healthy.

Post-mortem, in addition to the above changes, there were also found tuberculous meningitis and miliary tuberculosis of the lungs, tuberculous peritonitis with many adhesions, and matting together of intestines. Both the bronchial and mesenteric glands were enlarged and caseous.

The chief interest in the case appears to me to be that at no time whilst the patient was under observation in hospital were there any symptoms of intestinal or abdominal disease such as one would expect to find accompanying so marked changes found post-mortem.

For the clinical notes of the case my thanks are due to Dr. imThurm, House-Physician at the North-Eastern Children's Hospital, and to Dr. James Taylor I am indebted for permission to publish the case.

(*Exhibited November the* 18*th*, 1904.)

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29. CASE OF DOUBLE UNDESCENDED TESTIS.

By A. H. TUBBY, M.S.

Herbert U—, aged 3 years 7 months, is the second child of a family of four. There are no other abnormalities in the family. This child came under my notice at Westminster Hospital on September the 21st, 1904. At that time both testicles were in the inguinal canals. It was not thought advisable to operate, but rather to wait to see if any descent took place. The mother was told to attempt by gentle movements to push the testicles downwards. So far this appears to have been successful in the case of the right testis. The left testis remains in the same position as when

first seen, but the right is now outside the external ring. In the upper part of the right inguinal canal a hernia is appearing. (*Exhibited November the 18th,* 1904.)

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30. ORGANIC CAUSES OF SUDDEN AND UNEXPECTED DEATHS IN CHILDREN.

By CHARLES MACALISTER, M.D.

Sudden deaths among children, although, fortunately, of comparatively rare occurrence, are relatively large in number to the total number of sudden deaths at all ages, but the conditions which occasion them are frequently of so mysterious a nature that I am afraid that to whatever facts I may be able to give authoritative expression, it will be necessary for me to add some more or less hypothetical explanations to account for them. It is owing, no doubt, to the obscurity of their nature that a discussion has been invited at this special meeting, and one hopes that the experiences which will be elicited from gentlemen representing workers, both in the clinical and in the pathological departments of medicine, will greatly help to consolidate and bring to a focus our knowledge of the subject.

I may perhaps be permitted at the very outset of my remarks to digress for one moment, and to direct attention to the fact that in children who are the subjects of organic disease, it sometimes happens, strange as it may appear, that death may be simulated, and I have had two experiences illustrating this fact. One of these occurred during a period of residency in a hospital, when a night-sister came and reported the death of a child, and asked permission to have the body removed to the mortuary. Fortunately, the suddenness of the supposed death so surprised me that I went immediately to inspect the body, and found the child presenting such pallor, with cold and wet

brow, eyes partly open, yet breathing so quietly, and the heart's action so feeble after a crisis, that the mistake did not greatly surprise me. But, although this patient recovered, there can be no doubt that children who are improperly nursed and nourished in the course of severe acute illnesses sometimes die suddenly after the crisis has been reached, or during the period of convalescence. They go out with the tide, owing to nervous exhaustion or to some degenerative changes in the heart-muscle which have led to its enfeeblement. Faintness and giddiness are very common symptoms in children who have been the subjects of acute diseases, even when they have been well cared for, and it is very noticeable in them sometimes that a pulse, which remains good while the patient is still and recumbent, becomes frequent and poor on even slight exertion, betokening a feeble and irritable heart. One could well understand the occurrence of a fatal syncope if such a case were not carefully tended and the strength maintained.

A study of the excellent Reports of the Registrar-General, unfortunately, is quite unable to help us to elucidate the causes of the sudden deaths under consideration on this occasion, because, for the most part, they have been certified as cases of diseases of various kinds, and, where inquests have been held, no doubt many of them have been registered as due to congested lungs, or to whatever obvious condition has been revealed by an autopsy, or by clinical examination if the child has been seen shortly before death.

In any chapter which has been written on this subject it has been customary to classify the cases as being ascribable to syncope or asphyxia, to pulmonary, gastro-intestinal, nervous, and other rarer causes; but, while recognising that these may be convenient headings under which to introduce the matter, it must also be recognised that sudden deaths among children are not infrequently due to certain toxic conditions, which it would be difficult to group under any particular system because they tend to

affect several, the fatal issue sometimes being put down to gastro-intestinal, at others to pulmonary or nervous, disorders, according to the symptoms which had prevailed beforehand.

My chief reference will be to cases of this class, which have been familiar to me during a considerable number of years in connection with some institutions devoted to the care and keeping of children in which I am interested.

In studying these cases one of the great difficulties to be contended with is the fact that the deaths may take place so suddenly and so unexpectedly, with such trifling premonitions, that the child is not seen by a medical man at all, as the following case well exemplifies.

Last December, while the boys of one of our Industrial Schools were having an entertainment, which had been preceded by a tea, consisting of boiled ham, with sponge cakes and bread and butter, one of the little lads was noticed to be coughing a good deal in a way which was described as being rather irritating than distressing. He had previously been quite well, and it was the fact that his cough disturbed the entertainment that directed attention to him. The governor gave him some sweets, which he sucked without relief, and at last he very cheerfully went to bed and took an apple and an orange with him to compensate for the disappointment of losing the fun. Half an hour later the under matron was passing through the dormitory, and noticing the lad in bed lying very still, she went up to him and, turning his head, found that he was dead. Some fluid escaped from his mouth. Only half an hour had elapsed since this boy left the hall, and in the meantime he had undressed and tucked himself into bed, the apple and the orange which he had taken with him being found under his pillow. I, unfortunately, was not notified of the post-mortem examination, which was made by my house-physician at the Royal Southern Hospital (by order of the coroner), whither the body was taken, but in his evidence at the inquest he ascribed the death to syncope. There was evidence of bronchial catarrh,

but there was nothing in his larynx or windpipe. The stomach was full, and he suggested that the death had been caused by a violent fit of coughing. I should have felt disposed to consider that this death had resulted from laryngeal spasm or from some respiratory obstruction causing asphyxia, but for the circumstance that other somewhat similar cases had occurred in the same and some other schools—so frequently, indeed, that they received the appellation of "rapidly fatal institutional diseases" in a Report (subsequently published as a Blue Book) which was furnished to the Home Office concerning them about eight years ago by Dr. Sidney Coupland. Little light was thrown upon them at that time, but the general impression was that they were pneumonic in character.

At the same institution on February the 13th, 1895, a boy was brought into the sick-ward who had complained of headache to his playmates during the day, and had also been sick. Towards evening, with some suddenness, he was seized with violent vomiting and had some diarrhœa. He died within three hours, after becoming in succession quite powerless, deeply livid, and unconscious. There were crepitations throughout both lungs, and the immediate cause of death was asphyxia.

At the post-mortem examination, which was held on the following day, beyond some pulmonary engorgement, nothing was found to explain his death. The temperature was not recorded.

On July the 7th, 1897, a boy, previously strong and healthy, complained of headache on going to bed at 8 p.m. At 10.30 p.m. the master in charge of the dormitory woke the boys to empty their bladders, theirs being a dormitory devoted to boys who occasionally wet their beds. This lad again complained of headache, which was evidently severe, because he cried about it. In the early morning of July the 8th he complained to one of his school-mates that he felt very weak and ill, and at 6.10 a.m. the ward monitor called the ward master, stating that he had found him kneeling by his bedside, trying to get into bed, but

so weak that he could not get on to his legs, and had not power to drag himself in with his arms. After lifting him into bed, he was seen to be breathing with difficulty, he became unable to speak, and appeared to be unconscious. It was observed that he had evacuated his bowels in bed during the night. He died at 7.40 a.m., cessation of the respiration anteceding that of the heart. The temperature before death was 101·6° F. He looked exactly as though he had been strangled or suffocated, the whole body being intensely cyanosed everywhere, the eyes congested, and the veins of the neck turgid. The post-mortem examination revealed a very small patch of pneumonic consolidation in one of the lungs, and both lungs were much congested, a dirty brown fluid exuding from their cut surfaces. The brain and other organs presented no marked abnormality. On November the 11th, 1897, four months after the death of the boy which I have just recorded, another lad, previously perfectly well, complained of sickness on going to bed. He was thought to have a bilious attack, and some simple medicine was given by the matron. He was sick several times during the night and had a little diarrhœa, but towards morning he was thought to be sleeping quietly, and the matron kept his breakfast warm for him. Since, however, he seemed to be sleeping rather too long, she presently went to rouse him, and found that he was dead. I was hastily summoned, and there he lay curled up on his side, livid and apparently suffocated, just as the other boy had been. The body was cold excepting the abdomen, and he had evidently been dead for several hours. The coroner (with a perverseness which is detrimental to scientific investigation, and especially in a case like this, which puzzled us and alarmed us for the health of the whole institution) did not grant a post-mortem examination, and we can only assert on clinical grounds that this and the former case were probably of the same nature, and due to the same cause.

I might multiply examples of this sudden and terribly fatal disease by quoting other cases which have occurred

in my own practice, or which have been reported to me from various sources. I had a very similar case in the School for the Deaf and Dumb in 1893, and many have come to my knowledge as having occurred in other schools, whether for boys or for girls.

In most of the cases it has not been possible to associate the illness of these children with any obvious cause. On one occasion a drain in the school-yard was open for the repair of some flaw, but the others were unaccountable; nothing could be found in the sanitary conditions so far as drains were concerned, and the food was not apparently at fault. But I found one circumstance which aroused my suspicions and led me to recommend a radical alteration in the school hygiene, the adoption of which has apparently been followed by beneficial results, not only with respect to sudden deaths, but also in the general health of the institution. I refer to the methods of bathing the children.

There were two hundred boys in the school in which the fatal cases which I have recited occurred, and once a week they got a bath, all being bathed on the same day in a plunge bath, containing about three thousand gallons of water which was heated to 90° F. by means of steam. The boys were bathed in batches, those suffering from skin or ear troubles constituting the last batch.

Imagine, gentlemen, the state of that water after being used by even a fourth of the total number of lads—the horribly poisonous organic matter which it would contain, not to speak of the micro-organisms. Drs. Ernest Glynn and J. C. Matthews, in an investigation which they made ("On Bacteria in Public Swimming Baths" *), estimated that each bather shed about six thousand million bacteria, and that the number of organisms in this bath of 3000 gallons would rise to ninety thousand per c.c.

The bathing arrangements were immediately altered and the boys are now bathed on three days a week, every lad being washed with soap and water before entering the plunge; and it is very suggestive that whereas the death

* Thompson, Yates, and Johnson, 'Laboratory Reports,' 1903.

rate in the school had during the previous five years averaged 2·8 per cent. and only one year had passed without some deaths, which were mostly put down to pneumonia, no deaths occurred for four years and none from diseases of the rapidly fatal type, until the one which I quoted as having occurred a year ago, the cause of which was never ascertained.

I suspect very strongly that the toxin which causes these sudden deaths poisons the respiratory centre, and that the effects upon the lungs, which are noted sometimes as being simply engorged and œdematous, at others as presenting perhaps a small pneumonic patch, are not necessarily primary causes of death at all.

I would like to pursue this subject by referring to some cases of toxæmia resulting from sewer-gas poisoning in schools, but since none of them were fatal, I fear that I must refrain from enlarging upon an interesting matter and proceed with the material for this discussion. One of the most distressing types of sudden death is that which occurs in infants having all the appearance of robust health, and although a rare event, not a few cases have been recorded. In 1894 Dr. Harris* of Birkenhead spoke at the Medical Institution in Liverpool, concerning the appalling suddenness of the death of a healthy child aged 5 months, a well cared for, well-nursed, closely looked after baby which had had no previous ailments. On the day of its death he had been sent for because the child had had a crying night and seemed in pain. Its temperature, pulse, and respirations were normal; the lungs, heart, and other viscera right; the abdomen slightly tender to touch, but without tumefaction; urine said to be scanty. Dr. Harris diagnosed intestinal irritation, and ordered a dose of castor-oil at once, and five drops of sweet spirits of nitre occasionally during the day. The bowels were copiously moved, and in the afternoon the child was reported as about himself again. At five minutes to six in the evening it was started with a bottle by its mother, and

* 'Liverpool Medico-Chirurgical Journal,' 1894.

left taking it on the head nurse's knee quite as usual. At two minutes to six the nurse noticed a change, signalled for the mother, who, coming up at once, saw that it was death.

There was no choking, cough, or struggle, and the face was quite pale. The post mortem revealed nothing wrong with the viscera. The brain was not examined. Dr. Harris said death was from the heart, there was not a trace of asphyxia in it, and he asks the question, "Why should a healthy heart stop without a moment's warning?" I think the only rational explanation of a case such as this is the one advanced by Dr. Buzzard about twenty-three years ago, when, in speaking of a rare case of infantile paralysis, in which the face on one side was implicated, he suggested that this affection of the facial muscles was uncommon, because if the nucleus of the seventh was involved, that of the vagus would probably be also affected, and sudden death result. One cannot here enter upon a dissertation concerning infantile paralysis, but the feeling is growing that it results from a toxin the nature and source of which are not at present understood. The fact that instances have been recorded where two children in a family have been simultaneously affected is suggestive, and it seems probable that we shall have to look for some flaw in nursery hygiene to explain this curious and selective poison.

These, gentlemen, are examples of what I mean by rapidly fatal diseases resulting directly from toxic influences, and no doubt there are many others which might be classed under this heading, although they are more evidently associated with infective processes, if not with organic changes in the viscera. We must not forget in this relation the rapidly fatal cases of scarlatina, where the patient dies overwhelmed with the poison, and that occasionally the same event occurs in measles, quite apart from the results of pulmonary involvement. I remember seeing such a case in an adult, a lady who caught the infection from her infant. She had only felt really ill for

about twenty-four hours, although she must have been suffering for a few days, because she was covered with rash. On my arrival at the house, she was just dying (having become suddenly worse) from hyperpyrexia, the temperature in the axilla being over 109° F.

In these septic fevers sudden death occasionally results from hæmorrhage from the stomach, which comes from small pin-point or pore-like ulcers in the mucosa which can be seen, if formalin be injected into the stomach immediately after death has taken place so as to prevent post-mortem digestion. These ulcers appear to be embolic, and are usually derived from some local septic focus. I have seen one example in a case of measles, complicated by septic epiphysitis with involvement of the knee-joint where this profuse and unexpected hæmorrhage from the stomach took place, and, being uncontrollable, was the cause of death.

There is another hæmorrhagic condition which is worthy of note here, because it is quite distinctly a cause of sudden and unexpected death in infants and children; I refer to those somewhat rare cases where after an illness of only a few hours, generally characterised by diarrhœa and vomiting, followed by marked lividity and collapse, and often by the appearance of petechiæ, or bruise-like markings in the skin, the child dies and the post-mortem examination reveals hæmorrhages into the suprarenal bodies; I have seen an instance of this disease, and several have been reported.* Perfectly healthy and robust children are apt to be attacked and die in the most unexpected way. The histories of their cases remind one of the toxic diseases which were related at the commencement of this paper, and no doubt they result from poisons of a similar type; indeed, I omitted to mention in connection with the second case which I quoted that almost immediately after the child died another lad had a feverish attack, accompanied

* *E.g.* "Some Cases of Hæmorrhage into the Skin and Suprarenal Capsules," by Percy S. Blaker, M.R.C.S., and Bernard E. G. Pasley, M.R.C.S., 'British Medical Journal,' 1901.

by headache and vomiting, and quickly followed by purpura and thrombosis in the veins of both legs, which rendered him ill for a prolonged period.

I am disposed to believe that some volatile sewer product which gravely poisons the blood may sometimes cause these types of disease, or some poisons of similar character derived from other organic sources.

Before leaving the subject of the toxic and septic diseases which are associated with hæmorrhage, I must mention one other case where, in a little boy convalescent from scarlatina, so well that he was able to sit up and play with his toys, life was speedily ended by bleeding which took place from a small sinus in the neck, which had resulted from a superficial incision into a periglandular abscess. Colliquative necrosis had taken place unsuspectedly into the carotid artery, and a tremendous hæmorrhage resulted which rendered him unconscious, and he subsequently died in consequence.*

Besides the sudden deaths which take place in scarlet fever and measles, I must simply mention in passing that similar events have taken place in typhoid fever in children, especially where the disease has been overlooked or mistaken for simple diarrhœa, or for some unimportant febrile disturbance. Dr. J. Pearson Irvine, in a paper which he published in the 'Lancet' of November, 1877, records a number of cases of sudden death in young children, most of whom were brought in dead to Charing Cross Hospital, and among them were instances in which the pathological appearances indicated that the children had been suffering from enteric fever. Another point which should always be inquired into when sudden dissolution takes place is the possibility of an antecedent sore throat or other indication of diphtheria. I well remember a boy friend of my own dying suddenly after making some effort when he was recovering from what was taken to be a trivial illness, which proved to have been a diphtheria which had occasioned a degeneration and dilatation of his heart-muscle.

* Recorded in 'Pendlebury Abstracts,' 1885.

Another striking case of the same description is recorded by Dr. George Carpenter, in Vol. IV of the 'Reports of the Society for the Study of Disease in Children,' pp. 127—131, where a girl, aged 7 years, died suddenly from an uncomplicated myocarditis which probably resulted from an undetected diphtheria.

Fulminating smallpox will rapidly kill children as it does adults. A boy was brought into the Stanley Hospital many years ago, when I was physician there, in a condition of severe illness, the nature of which was not quite apparent in the out-patient room, but he was sent up to a small ward for observation. The nurse who was attending to him left the room for a moment, and on her return was amazed to find that he had died quite suddenly. The house-physician, who alone had seen him, then discovered that there was a rash on the lower part of the abdomen, and he expressed the opinion that the case might be one of malignant scarlatina. No doubt it was an example of the early roseola distributed over the lower abdominal inguinal regions which is seen in some cases of severe smallpox, for thirteen days afterwards the nurse who had attended him and several others went down with that disease.

Varicella is another disease which, although so unimportant in general, has its serious aspects. Varicella gangrenosa is a very rare condition associated with necrosis of considerable cutaneous areas. I have seen three cases of it, one under the care of Dr. Ashby, when I was house-physician at the Children's Hospital in Pendlebury, and the two others in the out-patient room of the Liverpool Royal Southern Hospital; each of these children died quite suddenly of cardiac failure, the result probably of exhaustion. It seems only liable to occur in weakly children or in those who are already the subjects of tuberculosis.

I am afraid, gentlemen, that in permitting me to open this discussion, in the section which refers to organic diseases, you have afforded me so wide a scope that it

would weary you were I to deal with the subjects in a very exhaustive way. I have spoken of the toxic and septic diseases at some length because a series of them have come my way, and collective information is sometimes useful, and I shall leave others to relate their experiences regarding sudden deaths connected with the circulatory, respiratory, and the systemic organs, but in speaking of sudden deaths in children who are the subjects of diseases presenting well-marked structural changes in the organs, I shall be as brief as possible.

Heart-disease in children has not often in my experience tended to cause sudden death. Isolated cases have occurred, and they have generally been in cases of mitral disease. I cannot say that in any of the cases the death has been unexpected, because almost invariably the children have been bedridden and actually suffering from the results of the disease. There is one case, however, always in my memory which has impressed me with the necessity for carefully watching cardiac cases to which digitalis is being administered. It was a little girl, aged about 8 years, suffering from mitral disease, who had been under treatment for some time, and she had improved greatly. I visited her late one night when making my rounds as a house-physician, and while speaking to her, she suddenly cried out that she was dying, stood up in her cot, and fell down dead. At the post-mortem examination neither ante-mortem clotting nor embolism was discovered, but the ventricle was firmly contracted, and I believe that digitalis, which she had been taking for some time, was probably responsible for it. I have no notes of this case and am speaking from memory concerning it, but the tragic circumstances connected with her death impressed me in a way which renders my recollection of the facts very vivid. It is surprising that the myocarditis which is present to a greater or lesser degree in some cases of acute rheumatism does not cause sudden death in children. The heart affection hardly ever produces symptoms during the course of the fever, but I recently had one boy, aged

12 years, under my care, whose condition gave me great anxiety on account of the irregularity and enfeeblement of his heart's action, with corresponding weakness of the pulse, and impoverishment of the first sound. He was exceedingly breathless on the smallest exertion and tended to faint, so that he had to be kept carefully recumbent. I had no doubt that a serious myocarditis complicated an endocarditis which was manifestly present, but he recovered at last, so far as his immediate condition was concerned, permanent valvular disease resulting from the endocarditis. My impression is that myocarditis is a very dangerous condition and one which if extensive would be very liable to cause sudden death; but as I have said, it must be a rare cause, so far as its connection with acute rheumatism is concerned. In infants congenital heart disease is a distinct cause of sudden death, if not shortly after birth, within a few months, the symptoms preceding the death often being of a convulsive nature. In older children death is not usually sudden, and in consequence it is fairly common for it to take place in the hospital wards.

Regarding diseases of the respiratory organs, I do not propose to dwell upon the subject of laryngismus stridulus, which, being a spasmodic affection, may fairly be dealt with in connection with functional disorders causing sudden death. There are just three conditions to bear prominently in mind with reference to this system, viz. pulmonary collapse, pulmonary inflammation, and pleurisy. Collapse of the lung is a subject of considerable importance in connection with the so-called death from overlying, and there is little doubt that some cases which have been ascribed to carelessness and drunkenness have been due to acute congestive conditions with catarrhal secretion and collapse. Brouardel, in his excellent book on "Death and Sudden Death," directs attention to this point and to the necessity for carefully distinguishing these cases lest a mistake should give rise to the assumption that criminal neglect or intent had to do with the case. Infants in a moribund condition are not infrequently brought to the hospitals,

livid and gasping from this cause, after illnesses of only a few hours' duration. In wasting and exhausting diseases this same cause of sudden death in infants is of course common enough, but is not one of the unexpected events in these cases. In pleurisy the pain may be sufficient to cause collapse and death, but if an effusion takes place rapidly in a case of acute pleurisy the respiratory distress becomes intense, practically from the collapse of the lung. I recollect seeing a child which was dying after only a few hours' illness from a pleurisy complicating post-scarlatinal nephritis. On withdrawing a small effusion which had taken place, the relief was immediate and permanent.

Regarding pneumonia as a cause of sudden death, excepting in the catarrhal types, it is not usual even in young children, but it certainly occurs and may prove rapidly fatal.

I shall not take up time by dwelling upon the nervous, alimentary, urinary, and cutaneous conditions, nor yet the syphilitic disorders which occasion sudden death; they are mostly well known and I mention them only in case they may bring to mind among those present cases which may be recorded in connection with the subject in hand.

(*Read December the 16th*, 1904.)

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31. FUNCTIONAL CAUSES OF SUDDEN AND UNEXPECTED DEATH IN CHILDREN.

By J. PORTER PARKINSON, M.D.

When I was first asked to open a discussion on the functional causes of death in children, I accepted with much misgiving, and with a deep sense of my own ignorance, and of the difficulties of the subject. The border-line between what is known as organic disease and functional disease is marked purely by the limits of our own knowledge, and is constantly changing as that knowledge progresses. For example, less than twenty years ago tetanus was con-

sidered to be a functional disease, and it is only quite recently that the bacteriology of chorea has been discovered. There can be no doubt that all nervous phenomena are the results of organic changes in the nervous system, and that therefore, strictly speaking, functional causes of death are non-existent. Still, the term is a convenient one to include those causes in which a primary structural change in any of the nerve elements is as yet undiscovered.

Marasmus.—In these cases sudden death is of very common occurrence. There may be no evidence during the day or two preceding death that the child is in any way different to what it has been for a considerable time. The death often occurs during the night, and no exciting cause can be found. The temperature has, however, frequently been subnormal for some days beforehand, and this may have something to do with the cause of death. Death occurs, as a rule, from heart failure, and sometimes atelectasis is found at the necropsy; in other cases up to now nothing abnormal has been discovered.

Convulsions.—The cause of death in convulsions is usually asphyxia from spasm of the respiratory muscles and larynx, and the post-mortem appearances consist of engorgement of the lungs with dark fluid blood, and a similar engorgement of the veins on the surface and in the interior of the brain, together with punctiform or larger hæmorrhages, and with an excess of cerebro-spinal fluid, both within the ventricles and on the surface of the brain. The right side of the heart and the pulmonary arteries are also engorged with blood. The tongue is frequently held between the clenched teeth, or shows signs of having been bitten. There are usually signs of rickets present in the ends of the ribs and bones generally.

In exceptional cases the child may die in convulsions before asphyxia has taken place, no doubt through the nervous system, the preliminary nervous discharge having led to disturbance of the vital centres in the medulla. In these cases the post-mortem appearances resemble those of syncope.

The difficulty of assigning the true cause of death in asphyxia will be alluded to again in speaking of overlying.

Laryngismus stridulus is a rare cause of death and shows but few post-mortem appearances beyond those of asphyxia. Fortunately, as a rule there is a history of previous attacks of spasm of the glottis. There is an absence of any morbid appearances on the larynx, such as inflammation or membrane, and no foreign body is to be found in the air-passages.

Enlargement of the mediastinal glands must be looked for, as occasionally sudden death from spasm of the glottis occurs in these cases from interference with the vagus nerve. Some years ago I published the reports of two cases in which this condition was found at the necropsy. The left vagus was found displaced and flattened out by the pressure of enlarged tuberculous glands in the posterior mediastinum.*

Some time ago I was asked to assist at the post-mortem examination on a child, aged 18 months, which had probably died from this cause, the doctor under whose care the child had been being doubtful as to the cause of death. There were marked signs of rickets in the bones, the other appearances being those due to death from asphyxia.

Overlying is a common cause of death from asphyxia in children, it having been estimated that at least a thousand children die every year from this cause in the city of London alone. The usual signs of asphyxia are found, but in addition to engorgement of the lungs with blood, they are sodden and œdematous, a large amount of frothy fluid being easily squeezed out of them.

These appearances resemble almost exactly those cases of the acute congestive form of pneumonia in which death has occurred during the first day or two, for no consolidation is present and the lung can, as a rule, be completely inflated. The microscope alone can decide the question, as signs of inflammation of the air-vesicles can be discovered as early as twelve hours from the onset of the disease. There is great distension of all the small vessels, and extravasations

* 'The British Medical Journal,' 1898.

of blood beneath the pleura into and around the air-vesicles, but in addition the alveoli are crowded with red and white cells, and swollen and desquamated epithelial cells are also found. The bronchi also show the changes of catarrhal inflammation. All these evidences of commencing inflammation are absent in the cases of slow asphyxia due to overlying, and the œdema of the lungs distinguishes them from the more rapid asphyxia in death from convulsions.

Death from emotional causes.—In the unstable and partly developed nervous system of a child the emotions predominate over the other functions; and as it is well recognised that excessive emotions, such as fear, anger, joy, etc., may prove fatal in the adult, it is exceedingly likely that they may be causes of death in children. As Professor Gairdner says: "It is difficult in such cases to say whether spasm or paralysis of the heart is the cause of death; in them the lungs are anæmic, a condition of cardiac apnœa has been induced." It is suggested that after the preliminary violent palpitation of the heart, the first result of emotion, the nerve-force conveyed to the accelerators of the heart is exhausted and the unrestrained inhibitory action of the vagus stops the heart, or perhaps emotion may act equally on the vagus and sympathetic, and the stoppage of the heart occurs from the exhaustion following the excessive discharge of centres with which both are in relation. That the emotions act especially upon the medulla seems to be shown by the frequency in which vomiting follows mental shock. In a case I saw recently a lady who had heard suddenly of the unexpected death of a near relation retched continuously for more than twenty-four hours.

The post-mortem appearances are not always like the one mentioned above. In one case of a boy, aged 7 years, who died during the excitement of watching a fight between two gipsy lads, the brain and cord were absolutely normal to the naked eye, the lungs also normal, though slightly gorged with blood; the pericardium was healthy. The right auricle was much distended with dark grumous blood; the right ventricle contained some dark blood; the left auricle was

full of blood, but not so distended as the right; the left ventricle contained a little fluid blood; the cavæ and pulmonary veins were filled with fluid blood. The other organs were healthy, and there were no signs of injury or contusion on the body.

From time to time we hear of cases in which no other reason than emotional excitement can be assigned as the cause of death, so it must be allowed that such cases, though rare, undoubtedly exist.

Death after a few hours of high fever.—Holt records some cases in which a child in apparent health is seized with great prostation and high fever and dies in a few hours. Some of these cases are acute congestive pneumonia, shown by the microscopical appearances of the lungs; others are doubtless cases of malignant scarlet fever or epidemic meningitis which have proved fatal before any definite sign of organic disease has appeared.

Heart failure after diphtheria.—I am rather doubtful whether this ought to be included in the present section, but the early manifestations of this disease are sometimes so slightly marked that they escape observation, and cardiac failure may be the only after-symptom. Two cases in which a sudden heart failure was due to this cause were reported a year or two back, in which children apparently healthy died suddenly, in one case while digging in the sand on the seashore.

If a microscopic examination of the heart-muscle be made, evidences of myocarditis are frequently found; but to the naked eye there may be no evidence of disease beyond slight and variable degrees of cardiac dilatation, and if the history of diphtheria be not given we may be at a loss to determine the cause of death. A case of this kind was recently reported to this Society by Dr. George Carpenter. Sudden death from cardiac failure may occur in the course of diseases such as acute rheumatism without hyperpyrexia or any discernible injury to the lungs or heart. Cases of this kind are alluded to by Sir W. Church in 'Allbutt's System of Medicine,' and a case was described

by Dr. Herringham before the Clinical Society in 1898, and also by Dr. Goodhart and others. In these cases the cause of death is probably myocarditis, which was shown to be present in Dr. Herringham's case.

The subject of shock as a cause of death will be dealt with in another section; but it is, of course, well known that death may occur at the onset of various diseases, such as cholera infantum, and other acute abdominal morbid states, before any marked evidences of toxæmia have appeared.

(*Read December 16th*, 1904).

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32. THE STATUS LYMPHATICUS AND SUDDEN AND UNEXPECTED DEATH IN CHILDREN.

By J. W. THOMSON WALKER, C.M.

The status lymphaticus has lately been credited with a large number of hitherto unexplained sudden deaths in childhood. The clinical picture presented by these cases shows three main groups.

(1) No cause could be assigned for the sudden death. In many of the cases there had been no warning that the child was not in its usual health. A child was put to bed and found dead. In some cases there was the possibility of "overlying," but in many this could be excluded. Again, a child might be suddenly taken ill while playing, and be hurried off to a hospital, but before reaching the hospital the child was dead. In some of these cases there were more or less pronounced premonitory symptoms. The child had been restless or disinclined for play, or for food, or had awakened from sleep shrieking, or had been observed to allow its head to fall back repeatedly. Some of these children had suffered from repeated attacks of difficulty of breathing, the so-called thymic asthma, and had succumbed during an attack.

(2) In a second group of cases the children were already

suffering from some disease. The disease might not in itself be sufficient to cause sudden death, as, for instance rickets, or the disease might be in itself an adequate cause of death, as in cases of tetanus (Blumer) and diphtheria (Dant).

(3) In a third group some definite cause of death could be assigned, but in most cases the cause was insufficient to cause death in a healthy individual. Such cases as death from a sudden plunge into cold water, intense excitement during card-playing, or sudden fear during a brawl in adults, and the injection of antidiphtheritic serum in a child had been followed by death.

In this group also are to be found certain cases of sudden death during or after anæsthesia, and also a certain number of stillborn children.

The manner in which death takes place in these children is extremely important. In the majority of cases in which death has been watched by a competent observer it has been ascribed to sudden circulatory failure or to simultaneous circulatory and respiratory failure. Almost all authors describe a marked degree of cyanosis, which has usually passed off rapidly, and been followed by pallor. Some observers have noted that the pulse and heart sounds suddenly ceased at the commencement of the attack. A small number of cases have undoubtedly shown evidence of asphyxia. Some cases have died during a convulsion which has been described as epileptiform.

The temperature has usually been normal, but in a few cases a rise of temperature has immediately preceded death, and in one of these it reached 105·2° F. half an hour after death. In the deaths during anæsthesia the pulse suddenly stopped, and the respirations, after becoming shallow, ceased. In deaths after operation the anæsthesia had frequently been quiet and uneventful, although in some cases the respirations had stopped and commenced again during the anæsthesia.

The post-mortem appearances were strikingly similar in these cases. The thymus was enlarged (persistent in adults)

and there was enlargement of the tonsils and the lymphoid tissue of the posterior one third of the tongue and of the pharynx. There was hyperplasia of the solitary follicles of the intestine, and of Peyer's patches. The spleen was moderately enlarged, and the mesenteric and thoracic lymph glands and some of the surface lymph glands, especially the axillary and cervical groups, were also enlarged. In addition the aorta was sometimes narrow and the heart small, and the genital organs were said to be small in some cases. The heart has been found contracted or dilated, the great veins full or empty. The blood was usually fluid. Petechiæ were frequently found in the pleural and pericardial surfaces. One authority has recently described microscopic changes in the lymphoid tissue which, he states, resemble those present in acute infections, but most authors state that the enlarged thymus and lymphoid tissues differ in no way from the normal.

Cultures from the blood and organs have almost invariably proved sterile. The condition has been found associated with certain other diseases and especially with enlargement of the thyroid gland, laryngismus stridulus, and rickets. In a fairly large number of cases in which death with the status lymphaticus has occurred during or after operation there has been enlargement of the thyroid gland. This enlargement occurs, however, in only a small percentage of the total cases of lymphatism, and the connection is probably accidental.

A number of cases of status lymphaticus suffer from rickets and from laryngismus stridulus, and the combination of the two latter conditions without the status lymphaticus is well known.

Some authorities hold that individuals suffering from the status lymphaticus are more susceptible to infections, and that the presence of this condition affects the course of the disease adversely. According to one author, more than one quarter of the cases of sudden death during diphtheria show the status lymphaticus.

The theories advanced to explain the phenomena in the

status lymphaticus may be divided into (1) pressure theories, and (2) toxic theories.

Endless discussion has surrounded the question as to whether pressure upon the trachea causes sudden death in these cases. Two facts are, however, indisputable. The trachea has only in the rarest instances been found compressed post mortem, and in the great majority of cases where death has been witnessed by a competent observer no signs of asphyxia were present.

In the small remaining number of cases death may be accepted as having been due to asphyxia.

Other theories have been advanced to show that the heart, the great vessels, the vagus, the phrenic or the recurrent laryngeal nerves, have been pressed upon, and death has thus been caused. The enlarged thymus is in a position to press upon any or all of these structures, so that arguments based upon anatomical considerations alone are valueless.

Numerous toxic theories have been advanced. Death has been supposed to result from the sudden over-production of an internal secretion from the thymus which may depress the heart or coagulate the blood. But no internal secretion has been proved to exist and the blood is usually found fluid after death.

Again, sudden death in these cases has been compared to that for sudden toxæmia in cases of summer diarrhœa (Thursfield). All the theories that are advanced are constructed to explain the cause of sudden death, but none of them refer to the cause for the enlargement or persistence of the thymus and the hyperplasia of the lymphoid tissue. It is accepted that the status lymphaticus is a congenital condition, but although a child may be born with the status lymphaticus fully developed, I see no reason to accept it as being invariably congenital. The analogy with other lymphoid hyperplasias leads one rather to consider the condition as a reaction to some chronic toxæmia, and this may also induce the hypersensitive state of the nervous system to slight shock which is present in these cases.

A few cases have been diagnosed or suspected during life from the discovery of hyperplasia of some part of the lymphatic system without adequate cause. In most cases, however, no reason for medical examination has been present before the sudden fatal result.

33. SURGICAL CAUSES OF SUDDEN AND UNEXPECTED DEATH IN CHILDREN.

By A. H. TUBBY, M.S.

When I was honoured by an invitation to open a discussion on this subject, I accepted it readily because of the opportunity it afforded me to be of some service to The Society for the Study of Disease in Children; and the subject is one which necessarily entails a considerable amount of reflection on records of cases; and it is a stimulant to inquiry as to whether those fatal results which have occurred suddenly can in future be avoided.

The title of the discussion appeared at first sight to be perfectly clear, and with very definite limitations, but when I came to examine more closely into its meaning, I was somewhat puzzled. Does it imply that the subject is to be treated under two headings, "sudden" and "unexpected" death, or is "suddenly unexpected death" meant, or "unexpectedly sudden death"? I have ventured to take the last named as the interpretation of the Society's wishes in treating this question.

Even from this point of view the matter is a wide and difficult one to place clearly before you. In a discussion which must necessarily range over the whole domain of surgery some systematic method of treating it must be found. With the ancients it was a doctrine that various organs might in different cases be called the *atria mortis* or portals of death, yet in practice we find that death is due to three causes: arrest of the heart and circulation, arrest of the respiration, and arrest of the action of the

brain. If, as the late Dr. Hilton Fagge pointed out, the only logical way of dealing with the question is to recognise those forms of death due immediately to conditions which lead directly to the extinction of life, we find that the last named cause, the action of the brain, is dependent upon the other two. And in sleep, the only obvious indications of life are the movements of the chest-walls and the action of the heart; thus we recognise that death—whether it be sudden or not—is ultimately due to one of these two conditions. There are, however, many avenues leading up to these portals, and in order to give what I hope may be a fairly intelligible account of the subject, I propose to discuss it under the following headings:

I. Circulatory Conditions.
II. Shock.
III. Toxic Conditions.
IV. Infective States.
V. Mechanical Causes.
VI. Conditions Markedly Referable to the Nervous System.
VII. Rare and Unusual Causes, not included in any of the above.

I. Circulatory Conditions.

These include: (*a*) abnormal conditions of the blood, whether states or diseases; (*b*) hæmorrhage, which may be not unfairly designated as spontaneous; (*c*) hæmorrhage after operation; (*d*) thrombotic and embolic conditions.

(*a*) Under Blood Conditions, whether States or Diseases, we must, beginning with the earliest days of life, speak of hæmophilia neonatorum. Congenital stricture or obliteration of the bile-ducts necessarily causes jaundice in infants. Some of the children succumb shortly after birth from persistent hæmorrhage from the umbilicus and intestinal canal; while those who survive may live a few months, giving time for hepatic cirrhosis to be established, which is ultimately fatal by intestinal and other hæmorrhages.

In other newly-born children hæmorrhages are due to congenital syphilis* or to pyæmic infection of the stump of the umbilical cord.†

True hæmophilia manifests itself very frequently in children, and a blow, an injury, or a simple operation may, on account of the loss of blood which it induces, give rise to fatal consequences. It is not necessary to recall instances where the extraction of a tooth, the removal of a tonsil, or a single tenotomy have resulted disastrously; but in those who are constantly dealing with the diseases of children, especially when calling for operation, it is of the utmost consequence to inquire into the family history when such cases are presented to one for treatment, as this important point may be overlooked. It has often happened that the parents have volunteered information that the child very "easily bruises," and this has put us on the *qui vive*, and probably saved us from disaster. In hæmophiliac children a slight blow on the head has given rise to hæmorrhage on the surface of the brain, or into the substance of the medulla, which is necessarily fatal.

With the onset of serious symptoms of infantile scurvy, or so-called scurvy rickets, wide-spread hæmorrhages manifest themselves with extreme suddenness. I can remember being called to see a child of the age of ten months, who had been brought up on patent foods, and presented an extremely yellow, cachectic appearance. He had had pain for four days at the lower end of the shaft of the left tibia, and was apparently going down hill rapidly. The diagnosis lay between subacute periostitis and infantile scurvy; but the temperature, instead of being raised, was subnormal. No other signs of hæmorrhage were present. An exploratory puncture settled the question. With very careful feeding, the child, although apparently dying at the time of being seen, ultimately made a good recovery. But, unfortunately, the course of the disease is not always so simple as this. Hæmorrhage may occur beneath the peri-

* Fichel, 'Archives f. Kinderheilkunde,' Bd. vii.

† Neumann, *ibid.*, Bänder xii, xiii.

osteum of many bones, or from the kidneys or alimentary canal, and from its volume and persistence may result in death. Even a localised hæmorrhage in vital spots, such as on the surface of the brain, has terminated thus.

(*b*) In discussing the subject, I take it we may allude to cases in which the condition, if not recognised, would certainly have led to suddenly unexpected death, and the object of such a discussion as this is to place more clearly before us the preventable causes. It therefore follows that those cases which go very near death, but are saved, must prove quite as instructive to us as those which are not. Under the heading of Spontaneous Hæmorrhage, it is allowable to include, in addition to those mentioned above, the cases which succumb from an ulcer in the stomach or duodenum, or from tubercular ulcers in the jejunum.* Umbilical hæmorrhage has already been alluded to, and its causes have been assigned as syphilis, pyæmia, and lesions of the bile-ducts and liver. Hæmorrhage from the genital organs is also due in newly-born children to these causes, or it is possible that it may be traceable to the presence of a calculus, and by its persistency may exhaust the infant. More formidable in older infants and children is a profuse flow of blood, due to sloughing of the pharynx and tonsils, a complication of scarlet fever of a severe type. Noma may spread to this region, and end in the same way.

(*c*) Fatal hæmorrhage in connection with and after operation is a somewhat sensational branch of our inquiry. It is preventable, and if it occurs must necessarily cause the surgeon the keenest anguish. Some want of thoroughness, even in the simplest operation, such as circumcision, in securing the frenal artery, has several times led to the sacrifice of the child's life. Bleeding occurring while under the anæsthetic is treated as oozing, and a little pressure is applied. The child recovers from the effects of the anæsthetic, either on its way home or at home,

* Cases recorded by Goodhart and Sawtell, Ashby and Wright, 'Diseases of Children,' 4th edition, p. 70.

bleeding starts afresh, and the parents, not recognising the dangerous effects of even a comparatively small loss of blood on infants' lives, hesitate or are unable to get assistance, and the child succumbs. It is on account of this susceptibility of a small baby to any loss of blood that the greatest care should be taken in hare-lip operations. More formidable and more difficult cases are those involving wide removal of tubercular glands in the neck, or of hygromata. It often happens that the glands surround closely the vessels in the carotid sheath, and I am acquainted with a case in which death followed profuse hæmorrhage, from wounding of the jugular vein near the base of the skull. In another instance a large hygroma was operated on in a child aged 2 years. The cyst had ramified in all directions, and presented extreme difficulties in its removal. During the operation much hæmorrhage occurred from the wounding of some large veins at the root of the neck. These were secured, and the child was placed back in bed, but he never rallied from the operation. I am inclined to think that some of these preventable deaths are due to what may be styled an attempt at brilliancy in operation, and personally I do not think that brilliancy in surgery is good for a patient. What is wanted is thoroughness and care, and attention to every minute detail which will contribute towards the successful termination of a case.

The introduction by Mr. Morant Baker of his india-rubber tracheotomy tube effected a revolution in the after-treatment of this operation. The prolonged retention of a metal tube is known to have caused ulcers of the trachea, and perforation of the innominate vein or artery. Later, in connection with tracheotomy, I shall have to allude to another cause of sudden death.

(*d*) Thrombosis and embolism are responsible for some tragic events, notably during the injection of nævi with perchloride of iron and other substances, or during electrolysis. There is a well-known case of nævus in the eyelid, which a surgeon elected to treat by the injection of a solu-

tion of perchloride of iron. Immediately after the injection the child gave a gasp, and fell back dead in its mother's arms. While dealing with the matter of thrombosis and embolism, their influence in determining a fatal result in the course of acute appendicitis or abdominal affections must not be overlooked, more especially as the clots themselves are septic, and are the source of metastatic deposits elsewhere, or even are the cause of the sudden development of brain abscess.

II. Shock.

It is quite true that a good many of the cases which are put down to shock should be more rightly ascribed to hæmorrhage, but, excluding such, we may discuss the question of shock under the following headings: (*a*) Cold, exposure, and undue delay in operating; (*b*) emptying cavities filled with fluid too rapidly; (*c*) washing out cavities.

(*a*) With reference to cold and exposure in operating, their effects are more particularly seen in operations for intussusception, intestinal obstruction, and laminectomy. An operation for intussusception should not occupy more than twenty minutes, and the utmost rapidity should be used in ascertaining the cause and if possible removing the source of intestinal obstruction. In a different connection I may quote a case of intussusception under the care of my colleague, Mr. Abbott, at the Evelina Hospital. The child was aged 3½ months, and the intussusception was reduced twenty-four hours after its onset. The child did well for one day, and then died suddenly. At the post mortem the thymus was found large, and very solid, and extending three quarters of the distance down the front of the pericardium. In laminectomy the exposure of the cord itself, particularly in the cervical and cervico-dorsal regions, is accompanied by profound nervous shock, and any aggravation of this by preventable exposure of the patient to cold is apt to be fatal. Attention to such small

details as wrapping the child's limbs and that part of the body outside the field of operation in cotton-wool and flannel, and having the operating table properly warmed by hot water, will, more often than is recognised, turn the scale in favour of the patient. More pointedly these remarks apply to operations on rickety children, on account of their liability to bronchitis and broncho-pneumonia.

(*b*) *Emptying cavities filled with fluid too rapidly.*—The observant anæsthetist will often report to the surgeon that at the moment of opening a pleural cavity filled with fluid or pus a distinct change takes place in the force and frequency of the pulse, which is lessened in both directions, and it has not seldom happened that we have been compelled to stop the flow of fluid while the equilibrium of the circulation is being restored. Precisely how this sudden change for the worse comes about we do not know, but arguing from the extensive displacement of the heart and large vessels which gradually occurs in these cases, any sudden restoration of these important parts to their normal position must embarrass their action. It may be that kinking of the large veins takes place, and the heart is deprived of its blood supply or the cause is a profound dilatation of the vessels inside the chest on account of the release from pressure. But whatever the cause may be, the shock is often profound, and taxes the energies of both surgeon and anæsthetist. The moral, of course, is obvious.

(*c*) *Washing out cavities.*—The remarks I am about to make on this point apply particularly to washing out the pleura and the cavities of large abscesses, such as psoas. Many years ago, when I was clinical assistant and house-physician to the late Dr. Moxon, he impressed upon us the danger of washing out the pleural cavity after evacuating an empyema. He recalled two cases of sudden death where this had happened. I can recall an almost fatal result in a patient under the care of a colleague of mine who evacuated several pints of pus from a psoas abscess. My colleague then proceeded to wash out the cavity with

some 1 in 40 carbolic acid solution. Immediately the fluid was injected the patient became blue and extremely collapsed. It is true that he did not die, but he was only just saved from death by the injection of strychnine and brandy, and lowering the head, and by a vigorous employment of artificial respiration.

Being myself much impressed by these cases, I undertook about seven years ago a series of investigations and experiments. These have only been published in brief in the 'British Medical Journal,' 1896, but I may here detail them more fully. The animals selected were dogs. Under the usual aseptic precautions an aperture was made into the chest, and the vertical limb of a Y-shaped glass tube inserted, and carefully tied in. To the oblique limbs india-rubber tubing and clamps were attached, so that it was possible to inject and withdraw fluids. It was found that if sterilised salt solution at a temperature of 99° F. were injected, a temporary and transient fall of slight character occurred, both in the blood-pressure and rate of respiration. Sterilised water produced a greater effect, boracic acid solution still more; while with even a small quantity of iodine solution (3j of tincture of iodine to the pint of water) the fall of blood-pressure was both rapid and great, and in some cases the animal succumbed at once, and in others it was only restored with difficulty by the rapid removal of the fluid from the pleural cavity and the employment of strychnine and artificial respiration. Most striking of all were the effects of a 1 in 40 solution of carbolic acid. This acted as certainly and as rapidly as a dose of hydrocyanic acid. The fall of blood-pressure was instantaneous and irrecoverable. It therefore seems that it is not the mere mechanical act of introducing fluid, but it is the chemical nature of the fluid, and this acts by reflex effect through the very wide and sensitive pleural surface upon the cardiac and vaso-motor centres in the medulla. In my opinion it is therefore better to abstain from washing out the pleural cavity.

III. Toxic Conditions which are responsible for Unexpectedly Sudden Death.

These may, from a surgical point of view, be discussed under the headings: (*a*) Chemical poisons and irritants; (*b*) toxic poisons.

(*a*) Of the chemical poisons and irritants most in common use we have carbolic acid and iodoform. With reference to carbolic acid, I strongly maintain that it should never be used as a compress to an infant's or child's skin before an operation. Most untoward results have followed its application. Intense erythema, vesication, sloughing of the skin, and even gangrene, have followed locally; while the absorptive power of a child's skin is such that fatal cases of carbolic poisoning and suppression of urine have ensued. It is, therefore, preferable, I find, to use a weak solution of biniodide of mercury (1 in 3000 or 4000), and not to have the compress on for more than two hours before operating. An infant's or child's skin has neither the horny character nor is it a receptacle for ingrained dirt as in the case of an adult, and it is therefore very readily cleansed. Certain infants are very susceptible to iodoform, and it appears to be more readily absorbed from some situations than from others. More particularly is this the case in the bladder, and the use of iodoform here has resulted in fatal absorption.

(*b*) *Toxic poisoning.*—In this connection it is my desire particularly to allude to some cases of sudden death in patients, especially children, who, according to the canons of our art, ought to have done well, after a timely and well-planned operation for appendicitis. I can recall at least three such cases which ended unexpectedly and suddenly to the great distress of the parents and the surgeon. One was that of a boy aged 13 years, whom I was asked to see in one of the suburbs of London. He had been ill for some days, and when I saw him there

were distinct signs of appendicitis, and indications of the presence of pus. I therefore operated, and found a thickened, but not perforated, appendix situated in a peritoneal pocket, with about half a teaspoonful of thick pus surrounding it. The pus was carefully sponged away, and the appendix removed, as it was quite easy to do so. The boy was much improved by the operation, and everything promised well, but sixty hours afterwards he attempted to raise himself in bed, and fell back dead. Another very similar case occurred in my practice last summer, except that the onset was more acute, and the septic symptoms were more pronounced. This child survived till the third day, and then, at nine in the morning, became suddenly collapsed, and died in five minutes. A third case was that of a young girl, who had had pain in the abdomen on a certain Friday, and went to bed on account of it. She was much better on the Saturday and Sunday, and on the Monday morning she asked for the daily paper. While sitting up in bed reading it, she suddenly fell back dead. In this case the appendix was found to have sloughed entirely. These are striking and lamentable instances of suddenly unexpected death, and they are difficult to explain. It is curious to note that in two of the cases the quantity of pus was small, and after its removal the patient seemed to be doing well until the fatal event occurred. It therefore seems that a poison of a particularly active nature, and of a cumulative effect—probably on the cardiac centres—develops and kills the patient. When I have had reason to suspect that such is likely to occur, or is occurring, I give directions that the patient shall be freely plied with digitalis, strychnine, and alcohol.

Uræmic poisoning is in children, as in adults, the cause of unexpected death after operations otherwise quite safe in themselves. Messrs. Ashby and Wright allude to a death after osteotomy, associated with granular kidney; and simple operations, such as for nævi or harelip, not seldom terminate suddenly from this cause. It is more easy to understand that the impaction of a calculus in a

child, giving rise to extravasation of urine, will, unless thoroughly and radically treated, result in death.

IV. Infective Conditions.

I propose to speak of the following diseases: tubercle, tetanus, acute œdema of the tissues of the neck, acute malignant œdema, and pyæmia.

As to tubercle, in several children's hospitals there are, I believe, notes of cases in which an operation has been done for the removal of a tuberculous lesion, and where, within two or three days, either acute tuberculosis or tubercular meningitis has developed. In looking over the notes of Mr. Abbott and myself at the Evelina Hospital for the past four years, I find two such cases, and it seems to me that such an untoward event is particularly likely to occur in operations on bone and the scraping of tubercular glands. In bone cases vessels which cannot contract are opened at the time of operation, and are therefore ready to receive direct implantation of tubercular material; and in gland cases the lymphatic channels are disturbed, and lymphatic thrombosis and embolus result. But I am anxious to allude further to another matter, and this, I fear, trenches somewhat upon the part in opening the discussion which has been allotted to my friend Dr. Blumfeld. In two instances which came under my observation, where a simple operation was performed for a non-tubercular lesion and signs of old phthisis were present in the lung, acute tuberculosis followed. Now, I observed that in both these cases gas and ether were given, and in both the anæsthetic was, so to speak, rushed into the patient. During the early part of the administration the patients became very congested and bluish-black. It therefore seems to me that I am not unreasonable in ascribing the death to the anæsthetic; and the history of events is probably that, in the struggling during the asphyxial stage of the anæsthetic, small portions of tubercular material were dislodged from the pulmonary arteries

or veins, and were freely distributed throughout the circulation.

Tetanus in new-born children, due to infection of the umbilical cord by dirty applications, is, fortunately, almost a dream of the past, and needs but a passing notice.

Acute œdemata present many points of interest, and I must allude in the first instance to that condition of acute inflammatory œdema of the neck which is designated by the name of angina Ludovici. It is particularly prone to occur after certain forms of tonsillar and pharyngeal inflammation, in association with perichondritis of the larynx, alveolar abscess of the jaw, and very rapidly follows the bursting of a suppurating gland beneath the deep fascia of the neck. In fact, there is no form of inflammatory œdema so explosive as that due to the last-named cause. Further, this œdema is frequently complicated by œdema of the glottis. Some of us have doubtless watched these cases very closely, and have been in constant attendance to perform laryngotomy or tracheotomy. A rare form of œdema is the acute malignant œdema of gangrenous emphysema which supervenes occasionally, especially in cases of fracture. I have seen two such cases, one which was almost fatal, and the other where the course of the disease was arrested early. The first case occurred in a young girl, aged 10 years, who fell down in a disused fowl-house, and sustained a compound fracture of the left forearm. The medical attendant, Dr. Southey Wright, very carefully cleansed the parts, and put the forearm up in splints. Twenty-eight hours afterwards he was called to the patient on account of extremely severe pain. He took the splints off, and examined the site of fracture. It was then found that an ichorous discharge was issuing from the wound, and the parts around looked white and dry. He administered an opiate, and wrapped the limb up in cotton-wool. The next morning he was called early, and found the hand was gangrenous and gaseous. By mid-day the limb was affected as far as the elbow. I arrived about two o'clock, and the gangrene

was extending to the shoulder. There was a large copper-coloured patch, the size of one's hand, on the left flank. Amputation was at once performed just below the shoulder, and as I happened to be acquainted with the fact that to the development of the bacillus of malignant œdema the presence of the Streptococcus pyogenes aureus is very favourable, I recommended the injection of antistreptococcic serum into the shoulder and the middle of the discoloured patch on the flank. A rapid improvement set in, and the patient recovered. The second case is that of a boy under my care at Westminster Hospital. He sustained a compound separation of the lower epiphysis of the left radius, due to the explosion of a firework. On the third day a like ichorous discharge and evil smell were noticed as in the preceding case. Antistreptococcic serum was at once injected, and the hand placed in a bath. The boy has now recovered. Cultures from the discharge developed gas.

Pyæmia in children is often rapidly and unusually fatal. In infants umbilical arteritis is often followed by general infection, and it not infrequently supervenes after lithotomy and lithotrity operations; but the most prolific source of pyæmia is acute necrosis of bone, where it is remarkable how soon after the onset of the disease general systemic infection takes place. I have known a child to die within forty-eight hours from the onset of the disease, with general infection.

Sometimes as a result of septic infection the simplest cases of operation assume the worst possible aspect. Thus, one case, in which an operation had been performed for adenoids, came under my notice, in which cellulitis of the neck followed, and was succeeded by double empyema, which caused the death of the child; and I also came across another case, in which a single empyema developed, but the child recovered.

V. Mechanical Conditions.

The entrance of foreign bodies into the air-passages. The immediate results depend upon the size and nature of the foreign body. If it be of such a nature, such as a large piece of meat, and of sufficient size to block at once the aperture of the glottis, the child does not exhibit the usual symptoms of asphyxia, but drops down as if shot, and forthwith dies. If, on the other hand, the offending substance be such as to partially block the laryngeal orifice, symptoms of asphyxia are extremely well marked. Unless the patient be eating at the time, and one of the bystanders be possessed of sufficient presence of mind to remove the foreign body from its situation, death may ensue without anybody being aware of the exact cause of it. In connection with laryngeal diseases, papillomata of the larynx may attain such a size and growth as to cause sudden asphyxia, owing partly to a valve-like action they have on the orifice of the larynx and the setting up of expiratory spasm.

The difficulties which occur in some children in inducing them to do without the tracheotomy tube are sometimes very serious. I recall the case of a small boy in Guy's, to whom we were frequently called, to rescue him from impending death, owing to laryngeal spasm coming on when the tube had been taken out in order to accustom him to breathing without its use. Eventually, by patience and perseverance, he was enabled to do so. I equally well recall another case at the same institution whom we restored several times by artificial respiration from a similar predicament, but who eventually succumbed to a spasm of this nature. And while speaking of affections of the respiratory tube I may allude to the sudden and often fatal dyspnœa caused by the pressure of a caseous gland upon the trachea or bronchi, and the effects of the bursting of such a gland into the lumen of those structures. The influence of a large thyroid and persistent

thymus have been dealt with by my predecessors in this discussion. But I may here mention the case of a boy, aged 15 years, the subject of exophthalmic goître, who was admitted into Westminster Hospital with the following history:

Enlargement of the thyroid gland began five or six weeks before admission, and he was treated with iodide of potash for a few days, and then sent home. He was readmitted on account of an attack of dyspnœa. Whilst waiting for operation he had a severe attack of this kind, and died before relief could be afforded. At the post-mortem examination œdema of the lungs was found, together with a large persistent thymus.

More or less mechanical conditions, dangerous to life, arise in connection with displacement of one or other of the cervical vertebræ. Such a case was under my care at the Evelina Hospital, in which, owing to congenital displacement of the fourth and fifth cervical vertebræ, whenever the child's head was allowed to be flexed forwards, alarming dyspnœa occurred. While it may be argued that the dyspnœa here was not entirely of a mechanical nature, yet no dispute can arise as to the cause of death in the following case: A small boy had been operated on for hare-lip, and, as the custom is, the hands had been tied down to the side, to prevent him from irritating the wound. At night this child was found lying prone with his face buried in the pillow, and the only appearances revealed by the post-mortem examination were those of asphyxia.

VI. Conditions referable to the Nervous System.

Instances of this will occur to you all. I have already alluded to hæmorrhage on the surface of the brain in hæmophilia, and I may here take the opportunity to remark on the effects of a hæmorrhage during the act of birth, arising from difficulties in the delivery of the head. It is comparatively seldom that the hæmorrhage is so extensive as

to cause death. Unfortunately, many of these children survive to become quite hopeless cases of infantile spastic paralysis or cerebral diplegia. The most usual cause of sudden death in children, arising from a brain lesion, is the bursting of a cerebral abscess into the lateral ventricles, or the rupture of a cerebellar abscess. Such cases are familiar to you, and I need not therefore give illustrations. The sudden escape of cerebro-spinal fluid is often responsible for a condition which is irrecoverable. On looking over various recorded cases, I find the notes of a child, the subject of cerebral abscess, which was successfully evacuated. At the dressing next day the gauze plug was removed, and with a pair of dressing forceps a second one was inserted, but in doing so there was a sudden escape of cerebro-spinal fluid. The child became collapsed, and died in a couple of hours. What had taken place is quite obvious—the lateral ventricle had been opened.

In the operation of laminectomy, if the cord be pressed very firmly against the laminæ, the attempt to insert even a probe beneath the bone and the cord was, in one of my cases, followed by such sudden collapse that I had to desist from the operation, resort to artificial respiration, and put the child back to bed. Subsequently we were able, by the application of vigorous stimulants before the cord was reached, to complete the operation successfully.

Speaking once more of the escape of cerebro-spinal fluid, reference may be made to a case of spina bifida, with a large nævus over it. The surgeon, not recognising the presence of a spina bifida under the nævus, attempted a rapid operation on the nævus, and excised it. Much cerebro-spinal fluid escaped, and the child died very shortly.

VII. Rare and Unusual Causes.

One of these is the sucking of air into veins in operation upon the neck for the removal of tubercular glands, and I may here remark that it may occur when the external

jugular vein is wounded, as well as when the internal or subclavian veins are injured. A case of nearly fatal results of wounding the external jugular vein during this operation came under my notice only last week.

And, finally, I may refer to the dangers of acute peritonitis arising from an inflammation of a retained testis in the abdomen, the presence of salpingitis in little children, associated with gonorrhœa and the sudden inflammation, suppuration, and bursting of a dermoid cyst.

These, then, gentlemen, are some of the causes of suddenly unexpected death in children. I am quite conscious of the sins of omission in dealing with the details of this very wide subject, but I am equally conscious that I have your indulgence in my imperfect attempt to bring the subject before you, and I trust I have raised such points as will evoke a discussion profitable to all of us.

(*Read December the 16th,* 1904.)

34. ANÆSTHETICS AS A CAUSE OF SUDDEN AND UNEXPECTED DEATH IN CHILDREN.

By J. BLUMFELD, M.D.

In discussing the question of death during anæsthesia in childhood I shall try, as far as possible, to avoid the overlapping which would so easily occur between my remarks and those dealing with the surgical aspect of sudden and unexpected deaths. For this reason the question of surgical shock, which as a matter of fact plays a large part in many of the cases which we ought to consider, will be, wherever possible, left on one side. It is necessary to state also, in order to clearly define the kind of case that should be here discussed, that I do not include any instances in which the patient's condition is serious before the administration of the anæs-

thetic. I propose, in fact, to limit my remarks to the discussion of cases in which a child succumbs during anæsthesia who was quite healthy except for the condition on account of which operation is undertaken, that condition not being one that has produced serious alteration in the child's general health. It is obvious that no more deplorable accident could be imagined than an utterly unexpected death in a healthy child undergoing a slight operation. And for this reason, if for no other, I think the Society is wise in discussing the question of children's deaths from anæsthetics as well as from purely surgical and medical causes. Moreover, these deaths are doubly terrible in that they are in most instances preventable. Yet they are far from uncommon, though their frequency can naturally enough not be gauged from the frequency of their publication. Yet, even with the natural reluctance that a practitioner would have in attributing the death of a healthy child solely to his anæsthetic administration, the Registrar-General's annual Reports furnish us with a number sufficiently large to show the real importance of the question. And it must be always borne in mind that these Reports, excellent though they are, probably greatly under-state the case. Without going into elaborate details, the figures very kindly furnished me by Dr. John Tatham show that in the four years 1899-1902 there were registered deaths in children under 10 years of age : From chloroform, 52 cases ; from ether and other anæsthetics, 18 cases ; in England and Wales (70 cases). In the five years 1899-1903 there were 59 deaths, nearly 12 in a year, from chloroform in children under 5 years of age only. These figures, which under-state the facts, and which largely represent avoidable calamities, are surely serious enough.

Now, there is one difficulty in adequately discussing this subject that at once presents itself. This is the fact, a fortunate enough one from other points of view, that any one individual anæsthetist's experience of such cases as we are concerned with is very small. Few of us happily can talk from any experience at all wide of unexpected deaths

in children under anæsthetics. The reason for this is, of course, that the majority of these deaths occur in the practice of junior officers of children's hospitals, and of practitioners who have comparatively rare opportunities for giving anæsthetics to infants and young children. And I may perhaps remark here that there is no class of case where a large experience is more necessary than in the case of infants. In no kind of subject is the difficulty of exactly estimating the patient's condition greater, nor is there any in which when a serious operation is to be performed the observation and skill of the anæsthetist is more severely taxed. This is an opinion which was formerly stated, too, by one of our Secretaries, Dr. Cautley, when, in conjunction with Mr. Clinton Dent, he discussed the subject of congenital pyloric stenosis at the Royal Medical and Chirurgical Society.

Now, the death of a healthy child during anæsthesia takes place apparently at one of three periods.

Firstly, the child may die before true anæsthesia is established and before any operation has been begun.

Secondly, death may take place in the course of an operation, anæsthesia being fully established, and the operation not being responsible by shock or hæmorrhage for the fatality.

Thirdly, death may take place at various intervals after the withdrawal of the anæsthetic.

With regard to the first class of case, in which the child dies before the anæsthesia is established, the explanation of the catastrophe is no simple matter. In the case of chloroform we are all familiar with the fact that the early stages before the advent of complete unconsciousness are the most dangerous. Exactly why this is so is not so certain. Two explanations are commonly offered. According to one the death is due to vagus inhibition of the heart, the vagus excitability being declared to be greatly enhanced during the early stage of chloroform inhalation. The other explanation declares that it is the temporary respiratory interruption due to spasm of muscles that is primarily responsible, this stoppage of breathing leading to dilatation

of the right side of the heart, imperfect elimination of chloroform from the lungs, and consequently continued action of the chloroform upon the musculature of the heart, which is already embarrassed, as we have seen by the respiratory interruption. A vicious circle entailing poisoning of the heart-muscle by chloroform is thus established. However the truth may be as regards the way in which these deaths are brought about, at any rate they occur with terrible suddenness. Moreover, there is as far as I know only one absolutely certain way of avoiding them. That way I shall allude to later. The remarkable feature about these cases is that they do not occur except in the case of chloroform. It might well be supposed that to suddenly and forcibly present a strong and irritating vapour to an infant's or young child's mouth for inhalation, and to keep it there, would be a proceeding likely to cause dangerous reflex cardiac effects, even if an actual asphyxial state did not supervene. Yet in the case, for instance, of ethyl chloride such an experiment is constantly being performed without untoward result, and the same may be said of ether. Such a proceeding is, of course, never adopted in the case of chloroform and would entail many deaths. And the cause of the early deaths that occur with chloroform cannot, I think, be always attributed to an approach to this sudden and air-excluding method of anæsthetising. It is not, I mean, at all clear that these early deaths are due simply to the exhibition of too strong a vapour. There are cases recorded occurring in experienced hands where it is as certain as such facts well can be that only a very dilute chloroform vapour has been offered for inhalation. Yet within a few breaths the patient has expired. The exact physiology of these syncopal deaths is not even now, in spite of much excellent, if highly controversial, research and writing, absolutely clear. The one fact that stands out both in the physiological laboratory and in the operating theatre is this, that it is with chloroform alone that these deaths occur, almost without exception. And this fact brings us to discuss the interesting question of the

part that fright and terror may play in bringing about fatal syncope before operation.

It is well known that in the pre-anæsthetic days patients died in this way before the knife had touched them, as well as with the shock of the first incision. The latter kind of shock we know to be avoided by proper anæsthetisation. But what about those who died of terror before they were touched? Why, it is said, should not patients die in this manner now? and are not some of these early deaths instances of fatal shock from mental emotion? It may be so; but we have to explain why such terror has never proved fatal when nitrous oxide or ether was the anæsthetic to be used. Whether children are more or less likely than adults to be unfavourably influenced towards the anæsthetic by fear is a point that the Society may well discuss. It is one, at any rate, both of practical and psychological interest. Young children are in their behaviour towards anæsthetics much more analogous with the lower animals than are grown-up people. The lower animals, and birds, I believe, in particular, are prone to die of fright. Therefore it might be argued that children are more likely to die in this way too. As a matter of fact, in practice it is more essential with a child even than with an adult to gain the patient's confidence and put him at ease and free from fear. The personal equation, in fact, in securing safe and satisfactory anæsthesia comes into play most markedly.

The *second* division we have made contains those instances in which death occurs from the anæsthetic during complete anæsthesia, when operation is proceeding; and obviously it might be argued that these are never purely anæsthetic deaths. When, however, the operation is one not involving important structures or large nerves or much hæmorrhage, when, in fact, it is what is called a minor operation, it is but fair to consider the anæsthetic responsible for the calamity. Now these deaths are, strictly speaking, never quite sudden. Their apparent suddenness is due to gradual failure of respiration or of pulse not being appreciated till the moment comes of absolute cessation, and the child is

thought to have suddenly died. These are the cases of which it may be said that they should never occur at all. They are the kind that never do occur to those who are constantly handling anæsthetics and children. The symptoms may, however, be so slight till danger is reached, and the child's behaviour under anæsthetics may be so misleading, that it is easy to understand their occurrence in other hands. These deaths are due to the direct toxic effects upon the vital centres of the anæsthetic drug used. In the case of ether it is, practically speaking, only possible to produce death in this way by the combination of some asphyxial influence; and the presence of this increasing asphyxia may not be readily obvious to the untrained.

The *third* kind of death is that which occurs after the anæsthetic has been withdrawn. When it takes place within a very few minutes, up to five, or perhaps even ten, for instance, the fatality in these cases is due to the continued action of the drug which, though inhaled some minutes before, had not yet been carried from the lungs by a failing circulation in sufficient quantity to exert its deleterious effects upon the heart or upon the medulla. In other instances of death after an anæsthetic and before the return of consciousness, the commencement of vomiting with inhalation by a still insensitive larynx of matter forced up into the naso-pharynx has been the direct cause. Such an accident occurs easily in the case of an infant who has been fed with milk too soon before an operation, the curd from the stomach proving the fatal body.

Nothing has so far been said as to whether there is any particular kind of patient in whom, though apparently healthy, death is more likely to occur than in others. Of course, there are conditions which, though not evident without special examination, are yet highly prejudicial from the point of view of anæsthetics. Deaths have resulted in such cases, and post-mortem examination has disclosed the contributory cause. Of this nature, for instance, was the death that happened under nitrous oxide in the case of a boy who had extensive mediastinal glandular disease with

actual pressure upon the trachea within the thorax. These cases, however, do not strictly come within the category we have laid down, because the patients are not in a state of ordinary health, although they may appear to be so to a superficial observation. Of an allied nature, perhaps, are the cases of the so-called "constitutio lymphatica," which some authorities consider so grave a condition in connection with anæsthetics.

Attention must be drawn to the fact that many of the deaths occur in connection with slight operations, particularly circumcision and removal of adenoids. These operations, of course, form a large proportion of the cases in which very young children undergo operation. Had time permitted, I should have liked to enter upon the question which is, after all, of the greatest practical importance—viz. that of the prevention of these catastrophes. I cannot, however, do more in this connection than suggest the following propositions for the consideration of the Society:

(1) That the opinion is too widely held and acted upon that in the very young chloroform is the only suitable anæsthetic.

(2) That in cases of circumcision, when complete unconsciousness has been obtained, the anæsthesia should afterwards be kept to a very light degree only. It is better that the nurse should have to control one thigh than that artificial respiration should become necessary.

I believe that even if these two simple indications were generally acted upon, a considerable decrease in the mortality of healthy infants under anæsthetics would at once result, "a consummation devoutly to be wished."

I thank the Society for the attention with which they have listened to remarks that are necessarily but incomplete, and that are likely to have appeared to many to border upon the commonplace.

(*Read December the* 16*th*, 1904.)

35. SUDDEN AND UNEXPECTED DEATH IN CHILDREN.

By A. J. ALLIOTT, M.D.

A FEMALE, aged 5½ years, went to school apparently well, but on coming home, 150 yards from the school (running), was seen to stagger and fall. She was carried into the house (about twenty yards off) unconscious, frothing at the mouth, and (?) "rattling in the throat." Medical help arrived almost immediately, but the child was dead (the mother thought she was dead before being taken into the house).

The mother stated that the child had seemed unwell during the previous two days, but that she (the child) was unwilling to have the doctor sent for. There was no history of rheumatic fever; she had had eczema a year or two previous, also she had had nose-bleeding during the two or three days preceding her death. There were no marks of injury on the body.

Post mortem fifty-six hours after death (long interval on account of the difficulty of obtaining the father's consent).

Heart.—Two drachms of fluid in the pericardium. On the anterior surface, just above the apex, was some yellow semi-solid lymph, adhering to the heart. All cavities empty. Ventricles strongly contracted. Valves healthy: no clot in the pulmonary artery close to the heart.

Lungs.—Healthy.

Brain.—No meningitis; cortex well developed and dark; puncta well marked. Whole substance of brain very soft and easily lacerable. In the right occipital lobe was found a softened and irregular patch about the size of a shilling, with ragged walls, but there was no pus or fluid of any sort in it; it looked as if the brain substance were deficient in amount. In the meninges—over this spot—was a small quantity of blood, not clot. (It was doubtful whether

this was pathological or not.) Elsewhere the brain was healthy, except in the fourth ventricle. In the floor of this, above and to the outer side of the auditory striæ, corresponding to the position of the nucleus of the pneumogastric, were two small hæmorrhages, one on each side, perfectly symmetrical. These did not extend deeply; they were not washed off, but were easily removed by the knife on cutting through them.

(*Read December the 16th,* 1904).

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36. SUDDEN AND UNEXPECTED DEATH IN CHILDREN.

By J. M. FORTESCUE-BRICKDALE, M.D.

Owing to the frequency of exploratory puncture of the chest in children, I think all cases of mischances connected with the operation should be recorded.

The patient was a boy, aged 2 years, who presented marked evidence of rachitis. He was admitted into hospital for pneumonia, there being dulness, bronchial breathing, and crepitating râles in the left chest as high as the angle of the scapula. At first he improved, but after the râles had disappeared the other physical signs persisted together with pyrexia. The child also had a sallow look, which suggested suppuration. A needle was therefore inserted in the seventh space, just outside the scapula. Nothing but blood was withdrawn, and the child, who was sitting up in bed supported by a nurse, became very restless, and almost immediately coughed up a little frothy blood. Cyanosis appeared, and the respirations became imperceptible. The child was inverted and a very little blood and mucus cleared out of the pharynx with the finger. Artificial respiration was then begun, and after about ten minutes the child made a few irregular gasps;

these, however, became weaker and further between, and in spite of subcutaneous stimulants he died. Artificial respiration was continued altogether for about two hours. Unfortunately, no post-mortem examination could be obtained.

I have found seven similar cases recorded, the first by Dr. George Carpenter, in 1893, in the 'American Journal of the Medical Sciences' (pp. 442-3). A child, aged 5 years, with cirrhosis of the lung, was explored. She coughed after the needle was inserted, blood began to pour from her mouth and nostrils, and she quickly died asphyxiated. Post mortem the stomach was found to be full of blood and the bronchi were filled with blood-stained mucus which extended on the right side as far as the medium-sized bronchi and on the affected side into the smallest tubes. Three cases have been reported by Dr. Thomas Oliver in the 'Lancet' (January the 2nd, 1904), and three by Dr. A. E. Russell in 'St. Thomas's Hospital Reports,' vol. xxviii. Four of these six cases were fatal, including two which occurred in children. Dr. Russell, who fully discusses the causation of this untoward accident, ascribes it to reflex inhibition of the cardiac and respiratory centres, due to irritation of some small branches of the vagus by the exploring needle. Brouardel, though not mentioning any cases of exploratory puncture for pleuritic effusion, quotes one in which the withdrawal of a few drops of blood in the attempt to aspirate a hydatid cyst was followed by sudden death. In this case the needle never actually entered the cyst.

A curious feature in one of the recorded cases is that the patient, after serious immediate symptoms, rallied, but died after a few hours. A similar history is given by Brouardel in the case of a man who received a blow in the epigastrium followed by collapse, from which he recovered, only to die during the ensuing night. Dr. Russell believes that a profound anæmia of the brain is the immediate cause of the inhibitory symptoms, and that when the fatal issue is delayed it is due to serious structural impairment of the brain-cells consequent on this anæmic condition.

It is unfortunate that there is no microscopical examination recorded of the vagus nerve. All the cases in the literature occurred in patients suffering from serious illness, and it seems highly probable, as Dr. Russell suggests, that a lowered condition of the nervous system is one of the causative factors. In a case of sudden death during a fit of coughing mentioned by Brouardel, in which miliary tuberculosis was found post mortem, compression and neuritis of the vagus, just above the cardiac branches, was demonstrated. This lesion, however, had not caused slowing of the pulse during life, and it is not quite easy to see how it affected the fatal issue.

(*Read December the 16th*, 1904.)

37. CASES OF SUDDEN AND UNEXPECTED DEATH IN CHILDREN.

By GEORGE CARPENTER, M.D.

For descriptive purposes I propose to divide the cases of this nature that have come under my notice into the following groups, though I am aware that objections may be raised to the classification of some of them, but this has been done for convenience of narration:

(1) Death from the heart.
(2) Death from the lungs.
(3) Death from the brain.
(4) Death following surgical operations
    - (*a*) without reference to the anæsthetic.
    - (*b*) with reference to the anæsthetic (delayed chloroform poisoning).
(5) Death from undetermined causes.

(1) Death from the Heart.

Of cases in which sudden or unexpected death occurred

from disease of the muscular walls of the heart, I have already recorded several examples in the 'Reports.'

Thus in a boy, aged 12 months,* who died suddenly and unexpectedly, extensive fibroid disease of the heart, which was not suspected during life, was found at the post-mortem examination.

In a girl, aged 8 years, who was admitted into hospital with mitral systolic and presystolic bruits and rheumatism, and who two months previous was perfectly well until she developed rheumatism, and subsequently chorea, death occurred quite suddenly while in hospital, and at the autopsy a fatty heart without valvular disease was found.†

In a girl, aged 9 years,‡ who was admitted into hospital suffering from a dilated heart unaccompanied by a bruit, but with a pulsating liver and a regurgitant pulse in the veins of the neck, the urgent symptoms abated under treatment, but subsequently suddenly the face became very blue, the eyes fixed, the limbs rigid, the pulse imperceptible at the wrist, and she died.

At the autopsy the heart was much enlarged, the valves were healthy, and interstitial myocarditis was found microscopically. The aorta was atheromatous. The cause for the myocarditis was not determined. Some infective condition was doubtless responsible for it.

In a girl, aged 7 years,§ and in whom there was neither history of diphtheria nor rheumatism, and who when brought to hospital had a feeble pulse, a weak first cardiac sound, and was somewhat cyanosed, together with a somewhat enlarged liver, urine loaded with albumin, absent patella reflexes, and a poorly acting soft palate, death occurred suddenly the following day. The heart was enlarged and very pale, and showed microscopically well-marked fatty degeneration of the muscle-fibres. The valves were healthy.

A well-nourished girl, aged 3 years, had whooping-cough

* 'Reports,' vol. iii, Plates I and II, pp. 177–179.

† 'Reports,' vol. iii, pp. 176, 177.

‡ 'Reports,' vol. iii, pp. 173–176, Fig. 20.

§ 'Reports,' vol. iv, pp. 127–131, Fig. 12.

in December, but she appeared perfectly well until the following July the 9th, when after eating a piece of bread she retched several times but did not vomit. During that day she appeared to be quite lively, and in the afternoon she went out for a walk with her mother. She had a good meal when out and a piece of cake before going to bed at 9.30 p.m In the small hours she was sick, but fell asleep afterwards until 7.30 a.m., when she was taken out to her grandmother's. On bringing her to hospital she lost consciousness on the way, and on her arrival there she was pulseless, breathed with difficulty, her temperature was 96·4° F., and she died soon afterwards. An exploratory puncture was made into her chest in two places, but no fluid was found.

Autopsy.—Over the lower part of the neck and spreading as far as the shoulder on either side and as low down as the xiphoid cartilage was a large plexus of dilated superficial veins. The abdominal cavity contained ʒiv of dark straw-coloured fluid, the right pleural cavity ʒvss, and the left ʒiij.

Right lung.—The lower lobe and the internal half of the right upper lobe were collapsed.

Left lung.—The bulk of the lower lobe was collapsed, and the upper lobe only partially crepitant.

Liver.—Fatty, yellow-ochre-coloured.

Gall-bladder.—Œdematous. Œdema extended to the tissues surrounding both kidneys and the whole length of the colon.

Mesenteric glands.—Enlarged and of a deep red colour. Spleen very firm to the touch. Malpighian corpuscles stand out plainly.

Kidneys.—Capsules strip easily. Stellate veins injected, exceedingly tough.

Heart.—Right side dilated, bun-shaped; valves quite healthy. A microscopical examination was not made.

A girl, aged 3 years, was weaned when 2 years old, and at 2¼ years she had "abscesses of the neck" and began to waste. She had diarrhœa on and off for a year, frothy

liquid motions; food passed undigested and cod-liver oil unaltered. On January the 25th she was taken into hospital and improved under treatment, so that the motions were less frequent, and she sat up in her cot and was lively. At 7 p.m. on February the 1st she was found cold and almost pulseless, and she died nine hours afterwards.

Autopsy.—Body emaciated. Œdema of the bases of the lungs. Large intestine thickened, and all along its course were ulcerations, with irregular edges and thickened bases. The mucous membrane was congested in the cæcal region, and thickened throughout. The mesenteric glands were much enlarged.

(2) Death from the Lungs.

Of sudden death from the lungs, one of the least known and by no means unimportant is that arising from exploratory puncture of the chest. In 1893, in the 'American Journal of the Medical Sciences,'* I drew attention to this danger.

The accident happened to a child suffering from cirrhosis of the lung. The case was sent into the wards of the Evelina Hospital, where the chest was explored by the resident medical officer. Immediately the child coughed, blood began to pour from the mouth and nose, and she died asphyxiated. Post mortem the lung was found to be cirrhosed. The stomach was full of blood, and there was some in the duodenum. The bronchi were filled with blood-stained mucus, which extended on the right side as far as the medium-sized bronchi and on the affected side into the smallest tubes. The larynx was full of frothy blood-stained mucus. Here death apparently occurred from suffocation, but fatal cases have been recorded by A. E. Russell† and Thomas Oliver‡ in which death was attributed to reflex inhibition of the respiratory and cardio-inhibitory centres, and was not attended by loss of blood.

* October, 1893, pp. 422, 423.

† 'St. Thomas's Hospital Reports,' vol. xxviii, 1898.

‡ 'Lancet,' January 2nd, 1904.

It does not apparently require a great loss of blood from the lungs to bring about a speedily fatal termination. In a boy, aged 5 years, who was suffering from whooping-cough and tuberculous broncho-pneumonia hæmoptysis occurred on August the 13th; ℥jss of bright red frothy blood was brought up. The child became livid, the lips blue, and the pulse, which was 160 to the minute, was very weak. On the morning of the 21st he coughed up ℥j of bright red frothy blood and was very pale afterwards. In the evening of that day after another fit of coughing he brought up ℥iij of blood, and died almost immediately afterwards from syncope.

Autopsy.—The right auricle was full of dark clotted blood, the right ventricle contained blood-clot. The left ventricle was empty. The lower lobe of the left lung was solid and tuberculous, and on section there were cavities which contained pus—the size of the largest being that of a split pea. Portions of this lobe were hard and fibrous to the feel. A small vessel was found, apparently ruptured, in one part of it. Coagulated blood in small amount was found in the bronchial tubes which also contained a puriform secretion. There were no cavities in the right lung. The bronchial tubes on this side contained blood which had been sucked in from the hæmorrhage in the left lung.

Another fatal case of hæmoptysis has also come under my notice. Thus a boy, aged 2¼ years, was in hospital from December the 11th to January the 3rd suffering from tuberculous broncho-pneumonia. On this latter date, at which time there were also meningeal symptoms, he coughed, "a great deal of bleeding" occurred from the mouth and nose, the pulse became imperceptible, and he died ten minutes later.

Autopsy.—General tuberculosis. In the right middle lobe of the lung there was a cavity the size of a large marble with a blood-clot in it.

In regard to an undetermined cause for death in empyema, the following is an example.

A boy, aged 4 years, was admitted into hospital on May the 3rd, with an empyema on the left side. On admission there

was considerable dyspnœa, the lips were blue, and the pulse at the wrist barely distinguishable. He was tapped with a trochar and cannula, and a drainage-tube was inserted. Pus ran freely to the extent of ℥x during the night, and the child was somewhat better the next day, but he died suddenly that evening.

Autopsy.—The left lung was bound down, airless, and covered with a thick coating of lymph. There was recent lymph on the anterior border of the right lung. The pericardium was adherent to the lung on the left side, and the heart was somewhat flabby. The other organs were normal.

(3) Death from the Brain.

A boy, aged 8 years, developed otorrhœa, subsequent to an attack of scarlet fever four years previously. He subsequently complained of slight headache at various times. Three weeks before admission into hospital he developed a mastoid abscess, which was twice treated by lancing, and at this time he complained greatly of headache. Three days before his admission the abscess was properly opened and drained, and during this period he complained more of headache, and was sick for the first time.

On admission into hospital he was a well-nourished lad, heavy and dull, but he sat up in bed. The pulse was 90 to the minute, irregular. Temperature 100·2° F. Both membrana tympani absent, ears full of pus. On the night of admission he was slightly sick once. He slept well but complained of headache. He suddenly became unconscious, and died twenty-one hours after admission.

Autopsy.—General meningitis. In the left temporo-sphenoidal lobe was an abscess the size of a pigeon's egg. This, which had burst, contained two teaspoonfuls of greenish stinking pus. The abscess communicated at its back part by an opening as large as a signet ring with the descending cornu of the lateral ventricle. Stinking pus was present in both lateral ventricles. The abscess wall was very thick. No thrombi were found in the sinuses. Thick pus was present in the internal ears.

A girl, aged 8½ years, was being treated for a second attack of chorea. *The heart-sounds were normal* and the choreic movements were extremely slight. She ate her dinner as usual, but at tea-time she was taken violently sick, and was said by the nurse to have plunged and struggled a good deal. When seen she was quite comatose, with marked rigidity of the right arm and leg, which were fully extended. The pupils were equal, but did not act to light. The face was pale and the breathing stertorous. She never rallied, and died two hours and a half afterwards. The urine was albuminous.

Autopsy.—In the left hemisphere of the brain was a large cavity filled with a currant-jelly-like clot. It lay completely outside the lateral ventricle. The anterior extremity communicated by a small rent just in front of the corpus striatum with the anterior horn of the left lateral ventricle. Through this opening blood had passed into the ventricle. A small quantity had escaped into the right ventricle through the foramen of Monro, and it had found its way to the third ventricle and *viâ* the iter into the fourth ventricle and as far as the cerebro-spinal opening. Source of the hæmorrhage was not detected. *Heart:* Vegetations limited to the edge and to the auricular aspect were found on the mitral valve. At one point a soft polypoid mass projected from the edge of the flap. *Kidneys*: Infarcts in both. *Spleen*: Large and soft; contained one infarct.

This case is an example of sudden death from cerebral hæmorrhage secondary to infective endocarditis in chorea, and is of added interest from the fact that heart disease was not suspected during life. But the following illustrations of unexpected death in chorea cannot be attributed to any coarse cerebral lesions. True, they were associated with trifling hyperpyrexia, but such cannot be held accountable for the deaths, which were probably toxæmic in nature.

A girl, aged 7 years, suffered from rheumatic pains for some six months prior to an attack of chorea, which developed fourteen days before she came under observation,

at which time the movements were general. The heart was normal. A fortnight later her temperature began to rise, and on October the 31st the temperature was 102·8° F. The movements at this time were very severe. From that time onwards she steadily got worse, was unable to speak, and could hardly swallow. She became more and more emaciated, the movements increasingly violent, and she died on November the 9th. During this period she twice suffered from epistaxis, and once was slightly sick, and the motions were loose. The temperature varied between 100° F. and 105·4° F., the latter being the highest recorded, and this occurred just before death.

Autopsy.—Heart: The edges of the mitral valve were roughened, and attached thereto were warty vegetations the size of a pea. The heart-substance was pale, and there were subserous petechiæ all over, especially over the posterior surface of the left ventricle. *Liver* swollen and marked by the ribs; substance pale, soft, and mottled. *Lungs* deeply congested, but crepitant throughout. *Kidneys* pale and soft; infarctions. *Spleen* normal. *Brain* normal to the naked eye.

In this case also the heart was thought to be normal during life.

A girl, aged 3½ years, was first seen on July the 12th. She had her first attack of rheumatism six weeks previous. She was well nourished. Choreic movements were general, but not violent. She had a mitral systolic bruit, rheumatic nodules on the back of her head, the dorsal surfaces of the hands, and about the ankles and feet. Under arsenic, in increasing doses up to ♏x thrice daily, the movements became much less. On August the 2nd she was wasted and her skin was very rough and dry. The mitral bruit was pronounced. She took food badly and was languid and drowsy. The nodes had disappeared. *Optic neuritis was discovered*, and her temperature at 6 p.m. had run up to 105·6° F. While under observation there had been some pyrexia, but 100·6° F. was the highest, and for several days prior to this large rise it had been more or less normal.

She was quite sensible when spoken to until 4 p.m., when she became delirious. The movements were excessive. She was unconscious all night and her abdomen was very distended. At 12 midnight the temperature was 106° F., and at 2 a.m. 106·2° F. Antifebrin was given earlier in the night, but without result. Powders were administered at 9·40 and 12 midnight; they produced great sweating at the back of the head, but nothing more. She was sponged at 2 a.m. without material benefit. The breathing at this time was rapid, the face livid, and the lips blue, and she died at 4.5 a.m. on the 3rd.

Autopsy.—The brain appeared perfectly healthy to the naked eye. The body was thin, but not emaciated. There was slight thickening of the mitral valve. *Lungs*: Slight bronchitis. *Liver* fatty, but not markedly so. *Spleen* normal. *Kidneys* normal. *Intestines*: Peyer's patches injected, together with the surrounding portion of the intestine; otherwise quite normal.

(4). Death Following Surgical Operations.

(*a*) *Without Reference to the Anæsthetic.*

While resident Medical Officer at the Evelina Hospital some years ago the following cases came under my notice.

A boy, aged 14 months, was admitted into the hospital with colourless face and lips, cold all over, and a very weak pulse, suffering from bleeding after circumcision. The operation was performed on December the 10th, and the bleeding was said to have continued until his admission on December the 12th. The hæmorrhage was stopped under chloroform, but the child remained cold and pale; he became extremely restless, and in spite of various remedies he died on the 14th, being slightly convulsed before his death. The temperature on admission was 97·8° F., and just before his death, some 36 hours afterwards, it had steadily risen to 104·2° F.

Two deaths occurred in sequence to tracheotomy.

A girl, aged 22 months, was tracheotomised for laryngeal diphtheria.

The isthmus of the thyroid was much enlarged, and as it was found impossible to open the trachea above it, the incision was prolonged downwards. Just as the trachea was exposed a vein was pricked at the lower angle of the wound. Profuse venous hæmorrhage followed, pressure was applied, and the bleeding ceased almost immediately. The trachea was opened, and the tube introduced, when the bleeding recommenced. The incision was enlarged and the vein was caught. Almost immediately after the child stopped breathing. Artificial respiration was resorted to, and the trachea was cleared with a feather, but after a few short gasps at intervals respiration ceased. Artificial respiration was kept up for about twenty minutes but without avail.

Autopsy.—A large vein was found coming off from the right anterior jugular, which was very large. The vein had been opened, and was found to communicate with the inferior thyroid plexus of veins, and then with the left innominate vein. The ligature on the bleeding point was found intact, lying on, but not encircling, the divided veins. *Heart*: Right auricle and ventricle filled with a small amount of frothy blood. Left ventricle also contained a small amount of frothy blood. Air was present in the left innominate vein.

A boy, aged 22 months, was tracheotomised for laryngeal diphtheria on October the 12th. On October the 16th he died suddenly when the outer tube was being changed.

Autopsy.—The wound in the trachea was not a straight cut, but forked, with the prongs downwards. This made a valve-like flap in the wind-pipe, so that very slight force was sufficient to fold it back into the interior of the trachea; indeed, its position was such that it was impossible to introduce a dilator or a tube without displacing it backwards to some extent. The heart muscle was normal Death occurred from asphyxia, and there was no visceral mischief.

(b) *With Reference to the Anæsthetic (Delayed Chloroform Poisoning).*

Of deaths following operations which were performed under carbolic spray, and for which chloroform was the anæsthetic, three examples came under my notice.

A girl, aged 4 years, with healthy organs and normal temperature, had her knee excised for pulpy disease by Sir H. G. Howse. She did not take the anæsthetic well, the breathing being "uncertain." She was not sick after the operation and slept during the night at short intervals. The following day at 3 p.m. the temperature had risen to 102·6° F. She was restless and fretful, but not sick. During the night she lay quiet, with her eyes partially closed, "starting occasionally, with twitching all over." Pulse very weak and irregular. Face and lips very pale. She was sick at 8.30 p.m., 12.15 a.m., and 4.30 a.m. From 6.30 a.m. she was slightly convulsed at times, and on and off unconscious all the morning. She was sick after a dose of champagne at 9 a.m., and again at 12 noon, when the vomit was coffee-coloured. At this time the temperature was 102·4° F. Subsequently she was sick immediately after taking food or medicine, and she was unconscious after noon and her face was rather blue. Subsequently the extremities became cold, and she died at 2.50 p.m., about 48 hours after the operation. *There was no autopsy.*

A healthy-looking and well-nourished boy, aged 3 years, with apparently normal organs and natural temperature, had his elbow-joint excised by Mr. R. C. Lucas, under chloroform, for pulpy disease. He recovered perfectly from the operation, but was very restless and fretful at night, crying out in his sleep. His temperature at 3 a.m. on the night of the operation was 103·8° F. The following day he was very quiet and drowsy, only taking notice when roused or spoken to. His face was pale, and he was very thirsty. The temperature at noon was 103° F. During the night he was very fretful and restless and he was sick

once, and three times after 7.30 a.m., and he died at 12 noon, the temperature after death being 105·4° F. For the relief of pain he was given small doses of laudanum.

An autopsy was performed (in August) 28 hours after death. The right auricle contained fluid blood and the right ventricle a blood-clot; the left cavities were empty. Muscular substance and valves normal. The lungs were crepitant and œdematous. The bronchial glands were enlarged and caseous all through. The left upper lobe contained two small old cavities, filled with a gritty caseous material. The mesenteric glands enlarged, but not caseous. Liver, fatty slightly. Kidneys pale, but otherwise normal.

A boy, aged 4½ years, was operated upon by Mr. R. C. Lucas for hip-disease under chloroform. There was no sickness at the time of operation. During the night of the operation he had very little sleep and was very sick once. The following day the temperature at 9 a.m. was 101·6° F. All that day he was very restless and cried out with pain. He was very sick at 8.30 a.m. and 4.30 p.m., and his face was livid at times. At 12 midnight the temperature was 101·6° F. He was very restless all through the following night; he had no sleep and suffered much pain. He was slightly sick twice and very sick once in the early morning hours. At 9 a.m. the temperature was 102° F., the pulse rate 140, and the respiration rate 40. All day long he cried out with pain, was rather drowsy at times, and at 3, 4, and 7.45 p.m. was very sick. At 9 p.m. the temperature was 97·8° F. He had no sleep on the night of this day, cried out as if in pain, was constantly sick, got suddenly worse at 3.15 a.m., when the temperature was 101·2° F., and died at 4.5 a.m. After the operation he had loose motions, as many as thirteen during one period of twenty-four hours.

Autopsy.—"All the organs were healthy." There was a false joint at the hip. A sinus led from the acetabulum directly into the bladder. The orifice of the sinus in the bladder was surrounded by small, pink, papillary excrescences and quite circular. The urine was normal.

(5) Death from Undetermined Causes.

The cases which I am about to relate probably all owed their origin to toxæmia.

In relation to the subject of delayed chloroform poisoning the following case is not without interest because chloroform was not taken and the symptoms were akin to those of delayed chloroform poisoning. A boy, aged $2\frac{1}{4}$ years, was admitted into hospital on July the 14th with the following history: He was always a weakly child. He attended as an out-patient and wasted very much, but then gained flesh, though it was said that vomiting and diarrhœa never quite ceased, but he was finally discharged cured. On July the 14th he was admitted because he was seriously ill. At this date he was a plump child and somewhat livid. There were no physical signs. Pulse rate 136, respiration rate 32, and temperature 99° F. July the 15th—Still no physical signs. Temperature between 101° F. and 102° F. Passed three loose motions. At 8 p.m. he became much worse, the temperature rising to 104·2° F., accompanied by severe sickness and delirium. The arms were moved about in a peculiar manner as if slowly convulsed, and he lay unconscious. The abdomen was retracted. The optic discs were perfectly normal. Sponging reduced the temperature several degrees, but by midnight it had risen again to 103·6° F. Sickness continued throughout the night, the convulsions and delirium (which became noisy) being very marked until 7 a.m., when the child died. The bowels were opened twice during the night and the motions were natural.

Autopsy (body packed in ice), twenty-eight hours after death. The *heart* was empty, the bases of the *lungs* congested. *Liver* very pale, *kidneys* also; neither unduly friable. *Spleen* normal; *intestines* and *mesenteric glands* normal. *Brain* healthy throughout. Longitudinal sinus filled by a loose and tolerably firm black clot, and connected with it were offshoots from the afferent veins on each side.

The anterior fontanelle was about the size of half a crown;

no other signs of rickets. The heat had been unusually oppressive at the time, and for some weeks prior to the child's death; but toxæmia, and not excessive heat, was the most likely cause for death.

From narrating this case I pass to another example of an unexplained death associated with jaundice, fatty degeneration of organs, and swollen Peyer's patches. A girl, aged 23 months, who had all but recovered from whooping-cough, had been slightly jaundiced for fourteen days and was languid, feverish, and constipated. The day before she was seen she appeared to be very feverish, and vomited a quantity of yellow fluid, and did not sleep much. On examination she was found to be well nourished and of a bright yellow colour. The liver was large and hard and reached nearly to the umbilicus. The spleen was normal. During the night she vomited bloody fluid three or four times; she became convulsed, and the pulse at the wrist was not perceptible. At 9 a.m. the temperature had risen to 101·8° F., and she died.

Autopsy.—Heart muscle somewhat pale. Under the microscope muscular tissue of the ventricles extremely fatty, not uniformly so, however, for fibres with normal striation were seen lying side by side with others in which the striæ were completely masked by oil granules. *Liver* large and hard, weighing 14 oz., markedly mottled, parts a dirty yellow hue, others a livid purple, passing gradually into each other; no softening. Under the microscope the liver-cells were extremely fatty. Cystic duct obliterated (? congenitally), gall-bladder full of bile-stained mucus. Hepatic ducts pervious and empty. *Pancreas* normal. *Spleen*, dark and firm, weighed $2\frac{1}{4}$ oz. *Kidneys* apparently healthy. Cortex somewhat pale. Capsule readily stripped. Under the microscope the convoluted tubes of the cortex were seen to be filled with oil. Urine not albuminous, depositing aggregates of rounded granules stained of a bright yellow colour. No leucin or tyrosin found in this or in the hepatic tissue. *Intestines*: Throughout the entire length of the small intestine from the upper part of the

jejunum to the ileo-cæcal valve the Peyer's patches presented a somewhat peculiar aspect. They were all of a dull purple colour, distinctly elevated above the surface. Seen from the peritoneal surface, their colour was observed to shine through, but no dilated vessels could be seen running across the bowels. The morbid change was not more advanced in one patch than in another, those highest up being in precisely the same condition as those at the lower end of the ileum. No signs of ulceration within the gut; no peritonitis outside it. Large intestine apparently healthy. In this case also toxæmia was probably the cause of death.

The following case is also of interest:

A girl, aged 4½ years, was admitted into hospital on April the 28th. Her illness was said to have commenced with a "fit" seven weeks previously, which left her paralysed, and since then she had wasted considerably. The attack began suddenly with drowsiness, followed in two days' time by frequent convulsive attacks, attended by foaming at the mouth, cyanosis, and struggling, extending over a period of ten days. Since the convulsions she had only spoken once, and had not walked or stood. Bed-sores appeared during the ten days' convulsive attack.

On examination, the child was extremely emaciated. She was unable to speak, and did not appear to recognise anything. There was no paralysis. The pupils contracted to light and were equal. There were large bed-sores over the sacrum, trochanters, the lower dorsal vertebræ, and the right shoulder. Elsewhere the skin was purpuric, and there was a large petechial patch over the back of the left hand. Urine and fæces were passed involuntarily. She could swallow liquids, and these only with difficulty. The following day (April the 29th) she developed emphysema of the face, both scapulæ, the sides of the neck, and the cheeks. By May the 1st the emphysema had extended to both eyelids and to the abdomen, and on May the 9th she died quietly and somewhat suddenly.

Autopsy.—Brain normal to the naked eye. *Lungs*: Slight

patches of pneumonia at the extreme bases posteriorly, along with hypostatic congestion and ecchymoses under the pleura. Nothing to account for the emphysema in the lungs or in the mediastinum. *Liver*: Very fatty. Organs normal otherwise. The air was found to extend over the scalp as high as the zygoma on each side and over the trunk as before mentioned. The limbs were free. The emphysema is difficult to explain. Gas-forming bacilli in the tissues are not likely to have been present. In this instance a microscopical examination of the brain would probably have revealed some alterations in the cortex.

A girl, aged 14 months,* enjoyed excellent health until she fell from a chair on the back of her head on June the 20th. She was sick after the fall, and twice the day following, when she was also drowsy. In the evening of June the 22nd she was thought to have had a blow on the ear, which was found to be red and blistered in the upper half. On the 24th she was comatose, face pale, extremities cold, pulse quick, pupils equal, reacting to light; respiration shallow and irregular, head retracted, temperature 102° F., slight clonic convulsions on both sides, fundus oculi normal. On the 25th the temperature at 6 p.m. was 104·8° F., and at 12 p.m. 106·6° F., when her pulse was very weak. On the 26th she was slightly sick once. The right extremities were quite hot and the left very cold; twitching was more marked on the left side of the body. The pulse was very weak. At noon the temperature was 107·6° F. The following night the pulse was frequently imperceptible at the wrist, the breathing laboured, the face twitching, and the extremities cold. She died at 10·45 a.m. on the 27th.

Autopsy.—Brain appeared normal to the naked eye. Patches of collapse in both lungs. Stomach stained with dark brown patches of blood on the mucous membrane. Pus in the middle ear, all organs otherwise free from disease.

* "A Case of Acute Middle-Ear Disease simulating Intra-Cranial Disease—Hyperpyrexia, 'Pediatrics,' vol. ii, pp. 430, 432.

No tubercle. Here also toxæmia was probably the cause of death.

(*Read December the 16th*, 1904.)

Discussion.

Dr. Leonard Guthrie proposed to confine his remarks to the question of unexpected or sudden death happening after rapid healing of certain cutaneous eruptions of a general character. First of all with regard to cases of syphilis. Fifteen years ago he had met with a series of cases of congenital syphilis in children 2 or 3 months old, which were brought up to hospital with widespread syphilitic eruption, snuffles, excoriations about the mouth and nose, and the usual desquamation of the palms of the hands and the soles of the feet. As he (Dr. Guthrie) was brought up in the belief that children took mercury well, he gave mercury, internally by means of hydrargyrum cum cretâ, and outwardly as an ointment to the soles of the feet, or applied to a bandage which was fastened round the abdomen or thighs. In a week or ten days the child would look extremely well, the eruption disappeared, and everyone was pleased with the result. At the next visit the mother would arrive at the hospital without the child and ask for a death certificate, stating that all had gone well, the neighbours admiring the rapid progress, but that afterwards the child became drowsy and vomited and died after convulsions. He had not the opportunity of making a post mortem in such cases, but he concluded that in all probability there was some syphilitic disease in the kidneys, and that death was due to uræmia set up by the action of the mercury in increasing the deficiency of those organs in elimination. At all events, after that experience he took to treating children very much more cautiously with mercury, and had continued to do so ever since. It was his practice now to give them doses of not more than $\frac{1}{4}$th grain of hydrargyrum cum cretâ in cases where there was a copious cutaneous eruption. Next, he would like to know if members had met with cases of chronic eczema of long-standing character which had been followed by rapid healing, and then very unexpected death. A child, aged 2 years, had had chronic eczema during most of its life. It was admitted into hospital and treated in a very simple way, though he did not remember how, probably by oxide of zinc ointment, and the child began at once to improve. In a week or ten days the eruption had nearly gone. The improvement was so remarkable that the matron drew the mother's attention to it, and the mother remarked "I feel sure you are curing that too quickly." The matron regarded it as an old wife's tale, but the next day the child had symptoms of gastro-enteritis and died. Post mortem

a marked condition of congestion was found in almost the whole gastro-intestinal tract. There were patches of injected arborising vessels, swollen and inflamed mucous membrane, and the Peyer's patches were raised above the surface and were dark red in colour. Another case was that of a child who was brought to the hospital with extensive ringworm of the scalp. An ointment, containing cantharide and red oxide of mercury, was prescribed, the mother being directed to rub small portions of it into small areas of the scalp at a time. But she plastered it over the whole head, and in twenty-four hours the patient died. He made a post-mortem examination and discovered hyperæmia of the brain and congestion of the kidneys, but nothing else. What was the explanation? He believed in some instances the death might be due to poisonous applications absorbed through the cutaneous surface, which was denuded of epithelium. But that would not account for all, and he was inclined to think there was some ground for the prevalent belief in the harmfulness of "driving in" an eruption, as it was called.

Mr. George Pernet had had a large experience of congenital syphilis in children, in the Skin Department of University College Hospital and elsewhere, and it was his custom to order 1 gr. of hydrargyrum cum cretâ three times a day; in some cases unguentum hydrargyri on the binder was prescribed. Yet as far as he could remember there had not been any instance of sudden and unexpected death. Had such occurred, application would have been made to the hospital for a death certificate, as such children were not usually attended by a practitioner outside. In congenital syphilis, of course, "snuffles" was a very important factor and required careful treating, for infants affected in this way tended to go down-hill rapidly from streptococcal infection and malnutrition. With reference to chronic eczema, the Skin Department of University College Hospital had to deal with many such cases in children, and efforts were certainly directed to curing the skin as quickly as possible. Many such cases had been in-patients, and he could not recall one which had died suddenly as a result of curing the morbid cutaneous condition. The status lymphaticus, about which they had heard a good deal recently, may have had something to say in the matter. He had certainly been told by old patients that when their weeping eczema was cured they did not feel so well in general health. He had felt there might be something in the contention of such patients. Perhaps the oozing surface gave the inadequate kidneys a rest and helped them in that way.

Mr. Arthur Edmunds drew attention to an experience of his own which he believed threw some light on the too rapid healing of cutaneous eruptions. The patient was a girl who had been severely burnt all over one side, producing an extensive granu-

lating surface, which was infected, causing serious constitutional disturbances. Under the hospital *régime* she improved. Then an attempt was made to skin-graft, the operations extending over a long period on account of the amount of surface involved. It was found repeatedly that if too large an area of skin were covered she would suddenly get very ill, with a temperature and a mild attack of septicæmia. The interpretation of that in his mind was that a skin-graft would be successful on a septic surface, though of course it was better to have few or no organisms there; that on a granulating surface there was free drainage of products, but the grafting corked them up and the drainage was checked. The absorption of the products was thus facilitated, with the consequent deterioration of health. In the cases of that kind, if a limited checking of free discharge could produce septicæmia, it was only a matter of degree for it to be capable of causing death.

Dr. Edmund Cautley desired to say a word as to the importance of the subject from the point of view of the general practitioner, for in most of the cases which died suddenly it was he who would be consulted during the acute illness or actually when the child was dead, and he would have to make his diagnosis either during the acute onset or at the post mortem. For that reason the subject deserved thorough ventilation. He would quote two cases which came under his notice as showing the difficulty of post-mortem diagnosis. One was a baby, aged 9 weeks, who had been suffering from wasting and improper feeding. He saw him on one day only, and death ensued early the next morning. The autopsy was made by a general practitioner, who found the intestines empty, the agminated glands above the ileo-cæcal valve distinctly inflamed, but no inflammation in the rest of either the small or the large intestine. The mesenteric glands were swollen and enlarged. The general practitioner, as the result of the autopsy, gave evidence at the inquest that the child died from "mesenteric disease," though he (Dr. Cautley) confessed he did not know what that was. The child was found dead in its "pram," by the side of the bed in the early morning, and it might have been from status lymphaticus. If more were known about that subject, it might be possible to diagnose it post mortem. The other case was a child, aged 9 months, the first-born, illegitimate, insured, and bottle-fed. None of those conditions were conducive to long life. He saw it in the Out-Patient Department, and apparently did not think it seriously ill, because he did not take it into the hospital. It died suddenly the same night at seven o'clock. The post-mortem examination was made by a general practitioner in the district, who found enlarged Peyer's patches and hæmorrhage into the stomach, and on that evidence ascribed death to typhoid fever. He thought this also might have been a case of status

lymphaticus. In neither of these cases was any evidence given as to the condition of the thymus gland. A more common type was that of death occurring suddenly in the course of marasmus, though the child was going on apparently perfectly well. The following was an instance. A child weighing 4 lbs. 10 oz., at 7 weeks old, had attained the weight of 10 lbs. 4 oz. at 7 months old. It had one fit ten weeks before death. Two days before death it had looked extremely well, and he had made a note to that effect. It gained 12 oz. during the week, though the diet had been the same during the last two weeks of life. The child was found dead in bed, with the face buried in the pillow. It was obvious that without careful examination post mortem one could not assign the cause of death, and even as a result of such examination one could not be certain. It might have been due to a fit, seeing that the child had had one ten weeks before. On the other hand, it might have been "overlaid," or have died from status lymphaticus. He also referred to the fact that the thymus was rarely examined in any such cases; seldom was there any mention of that gland in the post-mortem records—*i. e.* in cases which died outside the hospital. Even when they were examined by a special pathologist the state of the thymus was not always mentioned. The cases referred to by Dr. Macalister were interesting and of extreme importance; it was a condition which was recognised, but he (Dr. Cautley) had not encountered any such cases. He did not agree with Dr. Macalister that the bath water was certainly the cause of death in the cases mentioned, though probably the death was due to toxic causes: 90,000 microbes per cubic millimetre was nothing very startling. No doubt death was due to a toxine acting on the nerve-centre, but he could not subscribe to the view that the toxine was in the bath water.

The Chairman (**Dr. Frederick Taylor**) remarked that he had said earlier in the meeting that nearly everything in connection with the subject must have been exhausted, but one occasional cause of sudden death, at any rate amongst adults, had not been mentioned, namely, sudden death in chronic fatal diseases. Probably all had seen cases of patients extremely ill with a disease not necessarily immediately fatal, such as phthisis, who died suddenly without any adequate explanation of it. He supposed that there, as in many of the other cases, toxæmia might be the cause; one had thought of such cases living on the edge of a precipice and at length falling over, the falling over probably meaning an additional dose of the toxine which they had in their system day after day and month after month. When this occurred in the case of phthisis one naturally thought of pneumo-thorax and other contingencies, such as internal hæmorrhage, but after death nothing could be found in the body except the extensive lesions of the disease which were previously

known to be present. He had elsewhere recorded a case of phthisis which had been under his care at the Evelina Hospital for some time. The mother came for a certificate of death and stated that the child had suddenly died. He made a post mortem and found that the larynx was obstructed by a pea, which the child must have accidentally sucked into that position. The objects of medical efforts were cure and prevention. There was no cure for sudden death. The only case in which that had been touched on was one by Dr. Macalister, and there the death was not sudden. With regard to prevention, all members would like to review what had been said that evening, to see where prevention could come in. Of course, it was the business of the surgeon to prevent as far as possible the accidents which must sometimes occur in their practice, and the same must be said with regard to anæsthesia, which Dr. Blumfeld had fully discussed. But in many of the toxic cases it was much more difficult to see how the dangers could be prevented. It was a question of dosage, and that was a thing which the medical man could not measure. Probably the subject of lymphatism fell under the same heading, though he did not know that any detail had been forthcoming as to how lymphatism, or even an enlarged thymus, brought about sudden death.

38. TWO CASES OF VOLKMANN'S CONTRACTURE.

By FRANCIS JAFFREY.

Mr. Jaffrey showed two cases of Volkman's contracture.

Case 1.—H. S—, aged 2½ years, was first seen by him two months after the original accident. She fell off a chair and broke her right forearm. Splints were applied in the usual way (anterior and posterior), and two days later the limb was X-rayed, when the forearm appeared natural, though slightly swollen. On the fourth day a pressure sore appeared on the front and somewhat on the radial aspect of the forearm. Five weeks later it was noticed that the child could not open her hand.

When Mr. Jaffrey saw the patient, on August the 12th, 1904, there was a scar on the anterior and radial side of the right forearm. The fingers were strongly flexed, the

index finger being most affected. She could not extend her fingers and the wrist at the same time. The general nutrition of the forearm appeared good.

Case 2.—V. F—, aged 7 years, was brought to Mr. Jaffrey early in September, 1904, with a history of having fallen down a flight of stairs and breaking the right forearm. She was treated by the application of splints, and a few days later a pressure sore appeared on the centre of the forearm in front and another one on the dorsum over the wrist. Flexion of the fingers was noticed within a month of the accident.

When seen by him on January the 9th, 1905, there was marked atrophy of the muscles of the forearm and flexion of the fingers, the little finger being the least affected. The anterior pressure sore was not healed. The skin of the hand and fingers was somewhat glossy.

Both these cases are typical examples of Volkmann's contracture. Unfortunately, the electrical reactions have not been tried. The treatment adopted was massage and galvanism; the only improvement that was noticed was that the skin of the hand lost its glossy appearance in the second case. It was also observed in this case that there was a distinct arrest of the growth of the bones of the forearm. The chief interest in the cases now is as to what treatment should be adopted. Mr. Jaffrey suggested cutting down and lengthening the flexor tendons.

(*Exhibited January the 26th*, 1905).

Discussion.

The Chairman (**Dr. Percy Lewis**) (Folkestone) thought it would be well to carry out the procedure mentioned by Mr. Robert Jones, of Liverpool, in cases of infantile paralysis with contracture, namely, putting the fractured limbs in splints designed to stretch the contracted muscles, readjusting the splints two or three times a week, and increasing the tension of the muscles at each fresh application. The lengthened muscles were thus allowed to contract by never being stretched, while the shortened ones became elongated.

Dr. William Ewart said the treatment mentioned by Dr.

Lewis as having come from Liverpool had been applied by himself some years ago, when one of his house-physicians helped him to construct a small apparatus for a case of dropped wrist. It occurred to him that it was essential for a recovery of power in the over-stretched muscles to give them physiological rest from stretching by taking off the weight of the hand from the limb. An armlet was made with elastic traction so that the wrist was held up. This he believed to be the first indication in all treatment of partial paralysis of a limb where a set of muscles antagonised and overcame the feebler muscles opposed to them. The second principle of treatment was to afford the muscles a slight degree of exercise, beginning with passive movement; and the third to supply them with plenty of nourishment.

Mr. Donald Armour said a point often lost sight of was the necessity for taking down the splints and thoroughly cleaning the parts, not allowing the limb to remain more than three or four days to a week without being taken out. Certainly the splints should be removed at the end of the first forty-eight hours, and the limb thoroughly cleaned again before replacing the splints. The skin of fractured limbs in children ulcerated with surprising rapidity beneath badly applied splints.

Dr. Leonard Guthrie pointed out that in Mr. Jaffrey's second case there was a distinct difference in size between the two arms; the injured left forearm was much less developed than the right. He had a man under care, aged 50 years, who met with that accident and had that complaint at the age of 6 years, and the forearm remained at the size it must have been when the accident occurred. There had been considerable argument as to whether Volkmann's contracture was due to pressure on the nerves or on the vessels, or to myositis caused by pressure. Presumably it might be all three; more likely it was the circulation which stopped and almost killed the part. He thought the muscles were disorganised, and he doubted whether that could be recovered from, though the contractures and deformities might be remedied by operation.

Mr. Francis Jaffrey, in reply, said he would try the extension method mentioned by the Chairman. He had not previously had an instance of the condition to treat. The retardation of growth mentioned by Dr. Guthrie had been noticed, and that was why he (Mr. Jaffrey) suggested cutting down on the tendons and lengthening them. Cleaning the part frequently was essential in all fractures. Pressure sores were easily obtained, especially over the malleoli, where there was no negligence, because such parts easily became septic. They should be dressed with antiseptic gauze. He could not say whether there had been negligence in those particular cases, because he did not see them in their early stages. In the case of the smaller child the splint was taken off at the beginning of the fourth day, and even then the

skin had begun to slough, and the house-surgeon said it was not too tight.

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39. FIBROMA OF LARYNX.

By WILLIAM HENRY KELSON, M.D.

A. C—, aged 6 years, was brought to the London Throat Hospital complaining of loss of voice of three years' duration. There was no evidence of lung disease or hereditary syphilis, but the affection appeared to have followed an attack of measles. The hoarseness had recently increased.

On examination a pinkish growth was seen to be occupying the anterior commissure and right cord anteriorly, and this prevented the apposition of the vocal cords. The growth was removed through the mouth under chloroform, on recovering from which the voice was found to be clear.

Microscopically the growth was found to consist of fibrous tissue and dilated vessels.

(*Exhibited January the 20th*, 1905.)

40. LUPUS OF LARYNX.

By WILLIAM HENRY KELSON, M.D.

A. D—, aged 12 years, came complaining of loss of voice of a year's duration. There was no evidence of lung disease or hereditary syphilis, and no tubercle bacilli were found in the sputa.

On examination the upper part of the epiglottis was found to be wanting, and its stump was thickened and covered with red papules. The ventricular bands were similarly affected, as also the left vocal cord and anterior commissure: a portion was removed and the disease was

considered to be lupus. Neither arsenic nor anti-syphilitic remedies affected it, but marked improvement followed the intra-laryngeal application of the galvano-cautery, the voice returning.

Discussion.

The Chairman (Dr. Percy Lewis) (Folkestone) asked whether Dr. Kelson gave cocaine to the Lupus case or an anæsthetic.

Dr. W. H. Kelson, in reply, said part of the epiglottis of the Lupus case was removed under cocaine. The chief difficulty in the removal of laryngeal growths often resolved itself into that of properly cocainising the patient. He had tried eucaine, but without finding it at all satisfactory; it did not seem to produce the same absolute relaxation which was requisite when dealing with many laryngeal growths. Cocaine was also subsequently used for the cauterisations. There were never any œdema or other unsatisfactory symptoms following.

(*Exhibited January the 20th*, 1905).

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41. A CASE OF AORTIC DISEASE.

By LEONARD GUTHRIE, M.D.

Dr. Leonard Guthrie showed a girl, aged 12 years, who had been exhibited at the Society in December, 1903 ('Reports,' vol. iv, pp. 67–9), as a case of aortic stenosis. The left ventricle was hypertrophied, a systolic bruit was heard at the base of the heart, loudest over the pulmonary valves, conducted into the great vessels of the neck, audible faintly at the apex and in the axilla, and heard more intensely between the left scapula and spine. There was no aortic diastolic bruit. At the apex there was some semblance of a presystolic thrill and bruit, which he attributed to asymmetry of the two sides of the heart, and not to valvular mitral disease. He believed there was some con-

genital malformation in the neighbourhood of the aortic valves, but the girl was highly rheumatic.

(*Exhibited January the 20th*, 1905.)

Discussion.

Dr. C. W. Chapman said he agreed with Dr. Guthrie as to the aortic lesion, but not with regard to the mitral. He heard a presystolic murmur when the child was lying down. When sitting up he heard a divided first sound. He believed there was a lesion of both valves. He remembered seeing the case when last exhibited and discussing it.

Dr. Robert Hutchinson thought there was no doubt about the aortic stenosis from the position and character of the murmur. He agreed with Dr. Chapman that there was some mitral stenosis. He thought that it was not merely a reduplicated first sound, but an incipient bruit of mitral stenosis. It was known that a mitral stenosis murmur often took long to develop. If the case were seen at the end of still another year, he thought it would be found that there was then a distinct mitral stenotic murmur in addition to the aortic one.

Dr. Alexander Morison thought there was likely to be some pulmonary arterial disease in the present case from the booming character of the bruit, which was suggestive of a hæmic bruit. On examining the case that day when lying in the hospital he thought the thrill at the apex was dependent upon valvular disease of the mitral orifice. He believed it to be a developing constriction of the mitral orifice, not yet very complete; and it seemed to him that the more intense bruit found in the pulmonary area might possibly be due to the propagation of vibrations to the auricular appendix. There had been a good deal of controversy over the production of a bruit in the neighbourhood, and in this manner—the question was raised originally, he believed, by the late Dr. George Balfour.

Dr. William Ewart said that any opinion formed from the limited opportunities afforded when the case was exhibited was necessarily conditional, and those who examined the case quietly in more favourable circumstances could form a better opinion. Nevertheless he had succeeded in making the following definite observations. In the first place, there was dulness to the left of the manubrium, which was mentioned in some cases of persistent ductus arteriosus. Secondly, there was as high up as the first interspace considerable vascular excitement and loudness of the second sound, as if the pulmonary artery reached higher up than normal. Thirdly, when the patient sat up one could hear, with a rigid stethoscope, a distant rumble during the otherwise silent interval of diastole, which, of course, might be

of venous origin. He would be glad if the patient could be kept under observation and brought again later. He thought from the nature of the second sound that there was no disease of the aortic valves, but it was possible that there might be a constriction of the calibre of the aorta, though he found no evidence of it. The loudness of the murmur might perhaps be accounted for by some roughness of the surface of the aorta, but it was almost too loud for such a modest origin. He was still inclined to think that there might be some congenital defect of the pulmonary valves, that the ductus arteriosus might be patent, and that the proximal part of the pulmonary artery might be distended and thus give rise to the dulness mentioned.

Dr. Edmund Cautley said it was interesting that Dr. Guthrie should still be so confident after the lapse of a year, and as Dr. Guthrie had ample opportunities of examining the case one was loth to disagree with him. He agreed that there was a systolic murmur at the base, and thought that there was a short presystolic murmur at the apex—*i.e.* there was some contraction of the mitral valve. He thought there was a presystolic thrill there at times, and in addition a highly accentuated pulmonary second sound. If such a view were accepted, it would account to some extent for the character of the pulse. A pulse exaggerated on account of the hypertrophy might decrease in size from commencing mitral stenosis. The basic murmur varied a good deal with the position, and it was a possible view that the murmur was not intra-cardiac but due to the pulmonary artery being unduly in contact with the chest-wall. Against this view it might be urged that the murmur was not sufficiently localised. Dr. Guthrie laid considerable stress on the fact that the systolic murmur was louder behind on the right side than on the left, between the scapula and the spine, as a sign of aortic stenosis. He (Dr. Cautley) was not convinced that the murmurs of aortic stenosis, even in children, were heard better on that side. One often saw it stated in text-books that murmurs on that side were more frequently due to pulmonary or to mitral lesions. He would like to know on what evidence Dr. Guthrie based that statement, because it was a pity it should go out as a statement of fact from the Society unless there was strong evidence. He did not see why a different condition should be met with in children from that present in adults.

Dr. G. A. Sutherland believed the case to be one of a congenital cardiac lesion producing a double murmur of a humming character at the base, and probably caused by a patent ductus arteriosus. Dr. Guthrie said the heart was now in the same condition as it was twelve months ago, but he (Dr. Sutherland) did not recognise twelve months ago any mitral lesion, though this year there were evidences of such in the shape of a thrill and also a murmur at the apex. He did not agree with other

speakers about the timing of them. He believed both the thrill and the murmur to be systolic in character.

Dr. C. O. Hawthorne considered that the facts supported Dr. Guthrie's hypothesis. To his ear the murmur was purely systolic, and he judged both this and the exaggerated second sound to take origin in the aorta.

Dr. Leonard Guthrie, in reply, agreed there was nothing characteristic about the pulse; it was not particularly high or low tension. Dr. Cautley had suggested that he (Dr. Guthrie) was of the same opinion still; but he had varied, because last year he showed the case as one of aortic stenosis, whereas he did not think it was so now, because if so, by the present time there would probably have been signs of aortic regurgitation. He believed it to be some kind of congenital malformation, as Dr. Sutherland thought last year: possibly it was of the nature described by Dr. Theodore Fisher as commencing diaphragmatic obstruction in the neighbourhood of the valves of the aorta. He did not waver with regard to the mitral disease: he did not think such was present. He would gladly show the case again at some future time, when perhaps there would be as many different opinions as had already been expressed.

42. A CASE OF CONGENITAL MORBUS CORDIS.

By GEORGE CARPENTER, M.D.

The patient, a boy, aged $3\frac{1}{2}$ years, was brought to hospital in November, 1904, for jaundice of a month's duration, the attack being preceded by sickness and diarrhœa.

His father and mother are healthy; the latter had two miscarriages after his birth, and one child had died at a month old; there was no other evidence of syphilis.

The liver, which felt hard, was enlarged, its free edge being $3\frac{1}{2}$ fingers below the costal margin in the nipple line. The spleen, which was also hard to the touch, was two fingers in front of the costal margin. The cardiac area, as determined by percussion, was thought at that time not to extend to the right, but a skiagram which was taken subsequently showed that it extended somewhat beyond the

normal limits on that side. There was no epigastric pulsation. A loud systolic bruit, of greatest intensity over the third left interspace, half an inch outside the sternum, was audible all over the front of the chest, and was conducted into the great arteries of the neck and into the axillary arteries. The murmur was also audible in the back on the left side. A systolic thrill was felt all over the cardiac area. There was no cyanosis. The fingers were slightly clubbed. The diagnosis which had been made was stenosis of the pulmonary artery, associated with a perforate septum ventriculorum.

The liver and spleen are now normal in size. The condition of the heart and bruit are unaltered, but the child is now a little cyanosed, and the right side is now enlarged to percussion. He drew attention to the fact that enlargements of the liver are usual in catarrhal jaundice, and that in his experience about half these cases also developed splenic enlargements.

(*Exhibited January the 20th*, 1905.)

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43. A CASE OF AORTIC AND MITRAL DISEASE.

By ALEXANDER MORISON, M.D.

Dr. Alexander Morison showed a boy, aged 10 years, the subject of aortic and mitral valvular disease. There was an indefinite history of rheumatism, and he had suffered from pneumonia. The apex beat of the heart was in the sixth space and outside the left nipple line. There was a systolic bruit at the apex and traceable outwards to the left. There were also systolic and diastolic bruits in the aortic area, and the former was propagated into the arteries of the neck. The aortic diastolic bruit was traceable down the left edge of the sternum. The aortic systolic bruit was musical in timbre, and the point of maximum

intensity of the musical bruit was over the cartilages of the fourth and fifth ribs to the left of the sternum.

(*Exhibited January the 20th*, 1905.)

Discussion.

Dr. F. J. Poynton said that Dr. Morison's case was a good example of a very important form of heart disease in children, viz. combined aortic and mitral disease. So far as he knew, the prognosis of such cases was very bad, but he would like to hear other expressions of opinion. He had followed up ten or eleven of such cases, starting at about 6 or 7 years of age, and four of them had died, the survivors having at present severe symptoms. All children having aortic and mitral disease in early life seemed to have a serious future before them.

Dr. William Ewart thought the prognosis unfavourable in regard to this case, particularly if it happened that the temperature was slightly elevated. He founded that idea upon the musical nature of the systolic murmur, which suggested some pedunculated vegetation, or some torn condition of valve setting up vibrations, and would imply relative severity of the lesion. In cases such as this, with advanced mitral stenosis, coupled with advanced aortic valvular defect, causing a disappearance of the second sound of the aorta, no second sound was heard at the apex; and this demonstrated the fact that the pulmonary artery second sound, even when exaggerated, did not travel beyond a short distance at the surface of the chest, and had only a limited extent of conduction. That observation he had communicated some years ago in this same room, and he was still anxious to know whether it had been noticed before.

Dr. C. W. Chapman said the prognosis of such cases depended on the condition of health of the child at birth, and its environment since. If the children were fairly healthy to begin with, they lived a long time, especially if not over coddled, taking a fair amount of exercise. They generally broke down at about 17 years of age.

Dr. Alexander Morison, in reply, agreed that the prognosis in cases of double lesion was very bad, especially if there were any additional tethering of the heart by external adhesions. He had at present a patient, aged 49 years, with well-marked aortic regurgitant disease. He had rheumatic fever at the age of 9, and had done Trojan work as a labourer since. At 22 years of age he had smallpox. Recently he showed at the West London Hospital a man, aged 75 years, who had a well-marked constrictive murmur at the mitral orifice, attributed to rheumatic fever at 14 years of age. But in both these the lesion was a single one.

44. A CASE OF HYPOSPADIAS.

By DONALD J. ARMOUR, M.B.

The case shown to-day is a boy, aged 4 years, with a penoscrotal form of hypospadias. My object in showing him to the Society is twofold—first, to hear the opinion of the members of the Society as to the best age to undertake operation for remedying the malformation; secondly, to hear the views of those who have operated on these cases as to the most satisfactory method to employ.

(*Exhibited January the 20th*, 1905.)

Discussion.

The Chairman (Dr. Percy Lewis) (Folkestone) said he had such a case some years ago, and he did it by two stages. He left the opening as it was and made an anterior urethra, allowing the child to use the opening as before. He closed that after some weeks interval, first freshening the edges and bringing them together over an indiarubber tube. That was very successful, but it was the only one he had dealt with himself.

Mr. Donald Armour, in reply, said that the operation he did proved useless. The child had been circumcised before coming to the hospital, the defect not having been recognised, and the difficulty in micturition being supposed to be due to phimosis. By a modified flap operation he tried to create a new urethra, but though it was successful for a time, it broke down afterwards.

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45. A CASE OF HÆMOPHILIA HEREDITARY IN THE MALE LINE.

By ERIC PRITCHARD, M.D.

This case is one of hæmophilia in a girl, aged 6 years. She has suffered from very severe attacks of epistaxis since babyhood. She came to me for treatment in May last

suffering from subacute tonsillitis. She is a child of pronounced nervous type and of mental instability, occasionally giving way to bursts of uncontrollable laughter for no apparent reason. She shows no evidence of rheumatism, and has had no joint-lesions. She has been treated for the constitutional dyscrasia with five-grain doses of chloride of calcium, at first every night, and subsequently at occasional intervals. There has been no epistaxis since this treatment has been adopted.

The interest of the case centres in the hereditary history, which is unusual in two respects—firstly, in that the diathetic tendency to spontaneous hæmorrhage has been handed down in the male line for two generations, and in the third generation is evidenced in a severe form in the case of a female; the second point of interest lies in the fact that no other members of the family have, as far as I have been able to discover, suffered from symptoms of hæmophilia. The father of this child is subject to very severe attacks of epistaxis, and to very severe bleeding and bruising from slight injuries. He has had one attack, which was described as rheumatic fever, with serious joint trouble and cardiac complications. I have been unable to obtain any further particulars of his attack. The grandfather, who is still alive and only 50 years of age, similarly suffers from very severe attacks of epistaxis brought on by no apparent cause. He also bleeds readily and his skin readily bruises.

(*Exhibited January the 20th*, 1905.)

Discussion.

The Chairman (Dr. Percy Lewis) (Folkestone) asked how Dr. Pritchard dealt with the epistaxis, an important matter in hæmophilia. One of the most efficient ways he had found was to apply pressure from half an inch anterior to the line of junction of the cartilaginous with the bony part of the nose. As he used to be called up in the night to attend such cases, he had taught patients to stop it themselves. He recommended placing the arm over the patient's head, and hold the nose with the finger and thumb. After ten minutes it could be safely left.

Dr. Eric Pritchard, in reply, said he had not had occasion to treat the epistaxis. The child came for treatment for the

tonsillitis; he only learnt incidentally that she was a hæmophilic. He had made no observations on the blood, and there had been practically no epistaxis since. He had had two other cases of hæmophilia, both in adults, which had been practically cured by taking calcium chloride. It was more or less a general rule that when hæmophilics reached complete development there was a tendency for hæmophilia to disappear spontaneously, and it might have been so in the other two cases referred to.

46 A CASE OF CONGENITAL UNILATERAL PTOSIS.

By ERIC PRITCHARD, M.D.

This case was one of a boy, aged 14 years, who was recently brought to the Victoria Hospital for Children, suffering from severe attacks of migraine and more or less constant headache. He is a bright, intelligent lad, and takes considerable interest in work at school. The migrainous symptoms have become more severe since he has devoted himself more seriously to mental work. The migraine is presumably due to eye-strain and the effort of keeping the left eyelid (the one which is affected) raised by means of an exaggerated contraction of the frontalis muscle. There are no occular defects of any appreciable degree, and the movements of the eyeballs are normal and complete in all directions.

The defect was noticed a few weeks after birth, and at 15 months of age the boy was taken to the Moorfields Eye Hospital, where an operation was proposed, but not performed, owing to objection on the part of the parents. The ptosis of the left eyelid, which was apparently complete at birth, has remained in the same condition ever since without showing any signs of improvement. There is no associated spasm of the levator palpebræ in the affected lid when the left external pterygoid muscle is brought into action. An associated action of this kind, which was first described by Marcus Gunn in a case which he showed at the

Ophthalmological Society in 1883, and described in the 'Transactions' of the Society of the same year, has been noticed in some twenty subsequent cases reported by various authors. Inasmuch as unilateral congenital ptosis is a comparatively rare abnormality, the fact that associated spasm of the levator palpebræ when the external pterygoid has been brought into action has been reported in so considerable a number of cases of unilateral ptosis points to the fact that the absence of such an associated movement is the exception rather than the rule. The want of improvement in their case may be due to the fact that there is no nervous connection between the motor nucleus of the fifth nerve, which supplies the external pterygoid muscle, and the nucleus of the third nerve, which normally supplies the levator palpebræ. If this be so, it may explain both the want of improvement and the absence of the associated spasm. (*Exhibited January the 20th*, 1905.)

Discussion.

Dr. C. O. Hawthorne had seen several cases of congenital ptosis. The condition in most instances tended to improve, but that was not the case in Dr. Pritchard's patient. Another fact often seen with congenital ptosis was defect in the power of upward rotation of the globe, but here the ocular movements appeared normal. The defect in some of the cases was in all probability due to muscular abnormality.

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47. A CASE OF LICHEN PLANUS.

By GEORGE PERNET.

A boy, aged 6 years, in whom the disease commenced three months previously as the result of a kick on the left shin, followed by a fall, when he grazed it, according to the mother's account. The eruption commenced as pimples about the seat of the injury, and then rapidly spread to

other parts. When the patient was seen for the first time on January the 17th, 1905, there were numerous papules scattered about the trunk, most of them forming patches by coalescence, especially at the lower part of the back, where there was some pigmentation. The arms and legs were also affected in a similar way, and there were also some lesions scattered about the neck and a few situated in the forehead. The mouth was unaffected. The eruption was evidently involuting, but although only under treatment for a few days, there was already some further improvement, the eruption as a whole being less hyperæmic. The case was interesting, as Lichen planus was rare in such young children, and it was rarer still to observe it in them in the acutely developing generalised condition seen in this boy. A feature, too, of this case was the absence of itching, except now and then, which in adults was the symptom which, in widespread cases, usually dominated the scene.

The boy was interesting also from other points of view. He had been operated on for adenoids about a month before. He presented the skull deformity known as "tower skull" (*Thurmschädel*), and ptosis with absence of upward movements of the eyes. On the right side the ptosis had been corrected by Mr. Sydney Stephenson. The palate was highly arched.

(*Exhibited January the 20th*, 1905.)

Discussion.

The Chairman (Dr. Percy Lewis) (Folkestone) asked what were the prospects of the cure of the Lichen planus by salicin in the child.

Mr. Pernet, in reply, said salicin was useful in such cases, but there were other remedies.

48. A CASE OF ERYTHEMA MULTIFORME FOLLOWING VACCINATION.

By GEORGE PERNET.

A FEMALE child, aged 4 months, was vaccinated seventeen days previously in three places on the left arm; crusting present. Six days ago the eruption began on the chest. When first seen, three days previously, there were a number of circinate and gyrate lesions scattered about the trunk. They were about the diameter of a threepenny-piece, and had well-marked borders, with flattening of central parts. The child was poorly nourished and its stools were green and slimy. The latter may have played a part in the development of the rash. At any rate, treatment has been directed to disinfection of the alimentary tract. Erythematous and other rashes after vaccination were well known.*

(*Exhibited January the 20th*, 1905.)

Discussion.

Dr. C. W. Chapman, referring to the sequence of events in connection with vaccination, said that when in charge of a practice a lady, aged about 40 years, was to have been vaccinated, but owing to the sickness of a brother it could not be done. Four days afterwards she broke out with diffuse psoriasis. Had the vaccination been done, that would assuredly have been blamed for the condition.

49. MULTIPLE CONGENITAL OCCLUSIONS OF THE SMALL INTESTINE.†

By J. G. EMANUEL, M.D.

Dr. J. G. EMANUEL showed a specimen of congenital multiple occlusions of the small intestine from a seven months' infant admitted into the Children's Hospital, Birmingham,

* For a review of these see G. Pernet, "Vaccination Rashes and Complications," 'Lancet,' 1902.

† For a full report of this case with illustration see 'The British Journal of Children's Diseases,' vol. ii, pp. 457–462, October, 1905.

with the history of not having passed a motion and with having vomited since birth. As the result of an enema administered a large motion of meconium was passed. The vomiting continued till death occurred, on the ninth day.

At the autopsy there was found to be complete atresia of the bowel at the duodeno-jejunal juncture, and as a result of this the duodenum was greatly dilated and hypertrophied, possessing indeed a considerably larger volume than the stomach. In addition to this, the jejunum, in two other places along its course, was completely absent over a short distance of about half an inch, the mesentery at these places having a free edge, which was slightly thickened, but contained no lumen. Besides these three situations, in which there were complete gaps in the continuity of the bowel, there were five other points at which the lumen of the intestine (jejunum and upper part of the ileum) was completely occluded by a corresponding number of annular constrictions, so that the bowel immediately above these points was distended and filled with what appeared to be meconium, whilst that below was collapsed and empty. The lower part of the ileum and the large intestine were not affected in any way. There were no signs of intra-uterine peritonitis.

(*Exhibited January the 20th*, 1905.)

50. COLLAR STUD IMPACTED IN THE ŒSOPHAGUS AND PERFORATING THE TRACHEA.

By A. T. MARSHALL.

(Introduced.)

A female infant, aged 7 months, was brought to Paddington Green Children's Hospital on December the 28th with the following history: She had been under medical treatment for two months for a bad cough and frequent vomit-

ing. During the last three weeks both cough and vomiting had become worse, and she had taken food badly, while she was rapidly losing flesh.

On admission the child was very emaciated (weight 9 lbs.), was restless, and had an anxious expression. Temperature 98° F., pulse 124 to the minute, respirations 34 to the minute. There were dulness and bronchial breathing over the whole of the lower lobe of the right lung, with crepitations over the right apex. The left lung seemed normal.

While in hospital the child had a frequent cough, and there was always a quantity of mucus in the pharynx; no vomiting at all. She took food badly. Temperature varied from 96° F. to 102° F. Three days after admission she had a sudden attack of dyspnœa threatening asphyxiation. She was inverted, and about half an ounce of pus escaped from the mouth, after which the dyspnœa was completely relieved, and she took food better for twenty-four hours. She died, however, two days later with no fresh symptoms.

Post-mortem examination.—A metal collar stud was found impacted in the œsophagus, the flange of which had ulcerated into the trachea an inch above the bifurcation. The œsophagus was dilated above the stud and much constricted below. There was thin pus in both trachea and œsophagus. The right lung was completely consolidated, but there was no pus in the bronchioles. The left lung was healthy, except for a very small patch of consolidation in the lower lobe. The stomach and intestines were empty and collapsed.

At the inquest there was no evidence to show when or how the stud was swallowed.

(*Exhibited January the 20th*, 1905.)

51. CASE OF UNILATERAL CONGENITAL PARESIS OF THIRD AND FOURTH CRANIAL NERVES.

By N. BISHOP HARMAN, M.B.

M. H—, girl, aged 11 years. Mother dead, history defective. It is known the child was "born in a caul," so birth was presumably easy. The right eye is thought to have been "shut" since birth; no other members of the family are so affected. The muscles of the left eye and lid are normal; there is full and regular movement of the globe in all directions, the palpebral fissue is of normal breadth, and varies with the movements of the globe in the usual manner. V = $\frac{6}{18}$, Hm. 4D = $\frac{6}{12}$. Fundus normal. The right globe is hidden by the drooping lid, the lid is immobile except in so far as the vigorous contraction of the frontalis muscle can elevate it; the attempt is not very successful. On raising the lid with the finger the globe is found to be divergent about 30°; from this position it can be completely abducted, a movement proving the integrity of the external rectus and sixth nerve. But there is no other movement possible, either of deviation or rotation, so that the functions of all the recti, excepting the external and the two obliqui, and the nerves three and four supplying them, are lost. The orbicularis palpebrarum muscle is healthy and equal in power to that of the other side. The pupil measures 4·5 mm. in diameter; it is absolutely immobile, giving no reaction to light, convergence, or consensual stimulations. V. = $\frac{6}{60}$ Hm., 3·5D = $\frac{6}{36}$ one letter. There is no power of accommodation. Fundus normal. There are no other discoverable defects of body or mind.

To what this paresis of the third and fourth nerves may be due cannot certainly be determined; even speculation limps a little from want of a reliable "previous history." But it is clearly due either to disease or mal-development.

If disease be the cause, the lesion may have either a peripheral, a trunk, or a central location. Peripheral seems

excluded: the fourth and both divisions of the third with its intra-occular branches form too wide a group to sustain a peripheral lesion from which the neighbouring sixth nerve would escape. A trunk lesion is as difficult of support; in the cavernous sinus the first division of the fifth and the sixth are close to the paralysed nerves to escape the same damage, whilst at their roots the third and fourth are too far apart to be damaged by a single lesion.

The paralysis must be due to changes in the mid-brain stem, about the "*iter*." We have the choice of hæmorrhages or inflammations. Hæmorrhages about the wall of the iter might have affected these two nuclei, but to what cause are we to credit their production, seeing that birth may perhaps be taken to have been exceptionally easy? And we may wonder how such a grave lesion of the mid-brain affecting all of the several groups of nuclei constituting one third nucleus and a fourth nucleus could be consistent with a continuance of life. The most reasonable explanation seems to lie with the inflammatory hypothesis, some such lesion of the nuclei as we call "poliomyelitis," or "infantile paralysis"; similar processes affecting the cord are not infrequently found to select small groups of nuclei, and lead to atrophy of the muscles supplied by their axones. There is, unfortunately, no reliable history of the child's early years, so we are in the dark as to whether any febrile attack was associated with the onset of the paresis. A case of almost identical nature to the one now under discussion has been recorded by Silcock (1). In this case a healthy child had a febrile attack followed by complete and permanent third nerve paralysis of one eye, Silcock believed the cause to be "infantile paralysis" (no note is given of the condition of the superior oblique).

Any explanation of the paresis from defect of development is most difficult to support. These nuclei of the mid-brain belong to the oldest and most fixed part of the brain; anomalies, errors, additions, or omissions, in development are common enough in structures of late evolution, but almost never found in such ancient structures as these

nuclei. It is just as unlikely that the whole group of muscles should be absent from want of development. Individual muscles are not infrequently defective, "absent" so far as function is concerned, but in cases where anatomical investigations have been made an ill-developed and faultily placed muscular band has been demonstrated (2); the muscle was present but useless. In most such cases there are found irregular or abnormal movements of the eye. I have recently described such a case (3).

One point I would like to call attention to. The orbicularis palpebrarum muscles are well developed and efficient; the right is in every way as good as the left. If we conclude that this case is the result of a nuclear lesion, a conclusion practically certain, then by Mendel's hypothesis—that the orbicularis is an eye muscle receiving its primary innervation from the third nucleus—the muscle should be paralysed as well as the three recti, obliquus, and intra-ocular muscles; but the muscle is perfectly healthy. In some papers read before the Ophthalmological Society (4) I have brought forward a large amount of morphological and clinical evidence to show that the orbicularis is a proper facial muscle receiving its primary innervation from the facial nucleus. This case supports my contention.

References.

(1) 'Ophth. Soc. Trans,' vol. viii, p. 274.
(2) *Ibid.*, p. 272.
(3) *Ibid.*, vol. xxv.
(4) *Ibid.*, vols. xxiii, p. 357, xxiv.

(*Exhibited February the 17th*, 1905.)

Discussion.

Dr. Porter Parkinson said he thought there were two possible explanations of the condition, one of which had been spoken of as anterior poliomyelitis. In a condition of that sort one would have thought there would be some history of it, but he understood the mother was dead and no history could be obtained. A few meetings ago a case was shown by him in which the seventh nucleus only was affected. There was temporary hemiplegia at the onset, proving that the nucleus was the seat of the lesion. The other possible explanation would be a

hæmorrhage at birth. Those hæmorrhages were frequent enough in the substance of the brain, as reported by Dr. Spencer, who showed that in a large number of cases of stillborn children hæmorrhages were present in many parts of the brain, especially in the cerebellum. If it were so in the present case, one would hardly expect it to affect simply the nuclei of the third or fourth and not some of the other nuclei also. Therefore he inclined to the view that the cause was anterior poliomyelitis, or perhaps some congenital deficiency.

Mr. Sydney Stephenson regretted he had not heard Mr. Harman's account of the case. He inquired whether any heredity was traced, because a very striking point about such cases of congenital paralysis of ocular muscles was the influence of heredity. There was a well-known case on record by Gourfein,* where six members belonging to three generations of the same family all showed signs of third nerve paralysis. A few weeks ago he (Mr. Stephenson) saw a girl who had complete paralysis of the external branches of the third nerve on both sides, and possibly of the sixth, but not of the fourth. She belonged to a family of seven, four of whom showed a similar defect. The father of the girl was also affected. In his case, like that of Gourfein, five members were affected in two generations. The cause of such conditions was very obscure, and it was interesting to hear intra-uterine polio-encephalitis suggested as a cause. The usual explanation had been either a maldevelopment of the centres presiding over the movements, or defects of the muscles themselves. There was a certain amount of evidence on both sides. In the case he showed before the Society three years ago there were a good many points of resemblance to Mr. Bishop Harman's present one. There was almost complete paralysis of the third nerve on one side. It agreed with Mr. Bishop Harman's in another respect, viz. that the internal muscles of the eye, those presiding over the pupil and the function of accommodation, were also involved. As far as he knew, that was a very rare feature. But in that case it seemed very difficult to suppose that the defect affecting practically all branches of one third nerve could possibly be due to a maldevelopment, or absence, or imperfect insertion of the different muscles. It was much more likely to be due to some interference with the centre, which might be either in the nature of a diseased process such as Dr. Parkinson had suggested, or in the nature of an arrested development. One point in the case seemed to be in favour of the latter view, viz. that when the child was born she had ptosis, and during the first few months of life that ptosis became so much improved as to be scarcely perceptible when she came under care. There was also

* 'Arch. d'Ophtalmologie,' 1896.

the well-known case which was reported by Heubner, where a patient showed those complex paralyses, and died at the age of 1 year. At the autopsy it was found that there was a distinct atrophy of the bulb extending from the olivary body to the third pair. On the other hand, certainly in some of the cases the muscles implicated seemed to be absent or very small, as in cases reported by Henck, Steinheim, Baumgarten, Olbers, Liffenbach, and others.

Mr. N. Bishop Harman, in reply, said the only point which appeared to require an answer was Mr. Stephenson's question as to heredity. As he mentioned, the past history was very defective, owing to the mother being dead and the child being in charge of a relative who could state but little of the case. If he had heard that other members of the family had been similarly affected, he would perhaps have believed this case might be of congenital developmental origin. Failing that, he believed it must be due to some inflammatory process, such as that which was called "anterior poliomyelitis."

52. A CASE OF STILL'S TYPE OF CHRONIC JOINT DISEASE IN CHILDREN.

By F. PARKES WEBER, M.D.

The case is that of a girl, Rachel M—, aged 12½ years. Her mother is living and healthy; her father died of consumption. The articular trouble commenced during the winter 1902–3, in the left foot apparently. Soon afterwards the right foot was affected, and there was pain or stiffness in other joints. The hands were involved a few weeks later. The spinal column seems to have entirely escaped. In May, 1904, when I first saw her, there was chronic swelling about both wrists, with limitation of movement. No signs of cardiac or pulmonary disease were discovered. The edge of the spleen could be distinctly felt during inspiration below the costal margin, and there was slight enlargement of the lymphatic glands in both axillæ. The urine showed nothing abnormal. Fever was absent, and the child's general condition appeared fairly good. In December the condition was much the same. The axillary and inguinal

glands, but not the cervical, were moderately enlarged. So also were the tonsils. The splenic dulness was increased, and the edge could be felt at the costal margin with inspiration. The liver was probably of normal dimensions. Koch's old tuberculin, when the dose injected was five milligrammes, gave a definitely positive general reaction, the temperature rising to 101° F. after the injection. It must be noted, however, that the general reaction appeared not to be accompanied by any local reaction in the joints. At present (February, 1905) there is much limitation of movement in both wrists, the right one being the more swollen of the two. The general condition is fairly good. The tuberculin test has furnished a most valuable indication for treatment. Pure air and good food, which will best protect the patient from the development of active pulmonary tuberculosis, are agents likely to be also useful for the chronic joint trouble. She is shortly going to a seaside convalescent home or sanatorium.

In November, 1896, Dr. G. F. Still read a paper at the Royal Medical and Chirurgical Society on a "Form of Chronic Joint Disease in Children,"* previously confounded with rheumatoid arthritis, but to be distinguished by the accompanying enlargement of the spleen and lymphatic glands,† which is generally of moderate degree. The articular affection is multiple and symmetrical, without tendency to suppuration and without fibrillation of cartilage and osteophytic changes, but with capsular thickening and a good deal of swelling, probably due to effusion around,

* 'Med.-Chir. Trans.,' vol. lxxx, p. 47. See also Dr. Still's account of rheumatoid arthritis in children in Allbutt's 'System of Medicine,' 1897, vol. iii, p. 102.

† It is doubtful whether obvious enlargement of both spleen and lymphatic glands together should be considered as an essential diagnostic feature of this clinical group. Several years ago I observed a rheumatoid condition in the fingers and knees of a mentally deficient girl, aged 16 years, with moderate enlargement of the spleen, but I believe without any enlargement of lymphatic glands. The arthritic affection was quite chronic in her case, and probably a variety of Still's type occurring in an older patient.

rather than into, the joints. The hands, feet, knees, and cervical spine are, I think, most generally affected.

It is probable that Dr. Still took rather too gloomy a view of the prognosis, having at that time studied chiefly the graver cases, and that many relatively less severe cases of this symptom-complex occur, especially in somewhat older and less rachitic and cachectic children, in which great improvement, or ultimately even cure, results.

Owing to the occurrence of endocarditis in a case previously under my care,* and owing to the knowledge that pericarditis had been found in other cases, I was inclined to think that the whole symptom-complex in this clinical group of cases might be rheumatic, and I hear that Dr. Poynton has likewise observed a case in which typical valvular disease of the heart developed. Possibly the primary cause of the joint condition may vary in different cases, but, at any rate, I regard the hypothesis that in at least some of the cases it is ætiologically connected with tuberculosis as of extreme importance. A multiple symmetrical affection like this is not likely to be due to the local presence of tubercle bacilli in the affected joints, and the joints in these cases do not much resemble ordinary tuberculous joints. It is quite possible, however, that some cases are of the nature of what has been termed "tuberculous rheumatism"—that is to say, a symmetrical articular affection set up in specially constituted individuals by toxins from tuberculosis in other parts of the body, the joints, one may suppose, being picked out in these cases on the *locus minoris resistentiæ* principle. In the present case, owing to the positive reaction to tuberculin, there can really be no doubt that in some part of the body there is a not altogether obsolete tuberculosis, and the "tuberculous rheumatism" theory explains why after the tuberculin injection no local reaction was noticed in the joints. Edsall† gives the case

* See 'Med. Soc. Trans.,' vol. xxv, p. 344, and vol. xxvi, p. 346.

† 'Arch. Pediatr.,' March, 1904, p. 175. Dr. Ravenel demonstrated the presence of non-virulent bacilli, resembling tubercle bacilli, in the lymphatic glands of Edsall's case.

of a boy, aged 13 years, with Still's type of chronic arthritis, who reacted positively each time that the tuberculin test was tried; in this boy, it may be noted, joint pains accompanied the reaction. G. Mouriquand,* in his paper on "Tuberculous Rheumatism" in children,† quotes Poncet's view that tuberculosis, like many other infective diseases, may produce a condition resembling rheumatism.‡ In a case of Johanessen's, likewise quoted by Mouriquand, the patient, a girl, suffered from a rheumatism-like affection in both feet at the age of 4 years. Later on the elbows and knees, and afterwards the hands and other joints, were involved, and finally pulmonary tuberculosis led to death from hæmoptysis. (*Exhibited February the 17th*, 1905.)

53. A CASE OF CHRONIC ARTHRITIS.

By F. J. POYNTON, M.D.

The patient, aged 8 years, had suffered when an infant from rickets, and at 5 years had an attack of multiple arthritis, affecting the hands, knees, and feet.

There has never been quite complete recovery from this attack.

He is one of nine children; the others are healthy and the parents both living. There is no syphilitic history or rheumatic history, and the cause for the first attack obscure. No history of scarlet fever.

The boy is always pale, and has a poor appetite. The arthritis is much less evident in the knees than it was, but the hands, wrists, and feet remain much the same. At

* 'Gazette des Hôpitaux,' Paris, January the 21st, 1904, p. 69: abstract in 'Medical Review,' London, March, 1904, p. 154.

† On "Tuberculous Rheumatism," see also Mohr, 'Berliner Klinik,' November, 1904; and Barbier, 'Société Médicale des Hôpitaux,' April the 3rd, 1903.

‡ See A. Poncet, "Rhumatisme tuberculeux ou pseudo-rhumatisme d'origine bacillaire," 'Gazette des Hôpitaux,' July the 25th, 1901, p. 817.

intervals there are relapses, which last over many weeks, and during which the temperature is raised to about 100° F. At these times there is a good deal of pain in the joints.

The fingers have the spindle appearance of rheumatoid arthritis; there is some grating in some of the interphalangeal articulations. Over both wrists, on the dorsum of each, there is a circumscribed elastic swelling of the connective tissues.

The *axillary lymphatic glands* are enlarged and firm; this enlargement varies with the activity of the arthritis in the wrists.

The *spleen* has not been detected as enlarged. During the last year a systolic murmur has been repeatedly heard over the region of the impulse; it is faint and not traceable to the axilla. It is doubtful whether there is organic disease of the heart. The lungs are not affected. The disease has been most resistent to treatment. A radiograph showed no definite changes in the bones.

(*Exhibited February the 17th*, 1905).

Discussion.

The Chairman (Dr. Edmund Hobhouse) (Brighton) said he presumed the reaction to the tuberculin in Dr. Parkes Weber's case was a general one, and not affecting the local lesion. Such cases appeared to be of a para-tuberculous nature, occurring in tuberculous children, without giving rise to tuberculous joint lesions. Dr. Poynton's case did not appear to be of quite the same character. He did not glean that there was in that child any tuberculous history or suspicion of that disease.

Dr. F. J. Poynton replied that there was nothing obtainable in the history which was of any help towards the diagnosis.

Dr. F. Parkes Weber, in reply, said the reaction to tuberculin in his case was a general one, not noticeable in the joints. With regard to Dr. Poynton's other case, the one not shown at the meeting, it was very interesting that a definite organic valvular disease of the heart developed. Whether the murmur which had been heard in the boy exhibited by Dr. Poynton was organic or not one could not at present say, since it was not very obvious that evening. In his (Dr. Weber's) first case there could be no doubt that the boy at one time suffered from an actual attack of endocarditis, since which time there had been a persistent systolic mitral murmur.

54. OSTEOMYELITIS OF THE UPPER END OF THE FEMUR.

By EDMUND CAUTLEY, M.D.

On December the 16th, 1904, a boy, aged 9 years, was admitted into the Metropolitan Hospital under my care as a case of rheumatic fever. His temperature was 103° F., and he complained of pain in the left thigh. The pain had begun on the previous day. There was no family or past history indicative of rheumatism. On examining him two days later the only evidence of disease consisted of pain in the upper part of the thigh, some limitation of movement, though the joint did not seem to be involved, and fever, which had not yielded to the action of salicylate of sodium. A surgeon was asked to see him with a view to the case being one of early hip-disease, and he thought the joint was not affected.

Blood counts on December the 19th, 20th, and 21st showed a leucocytosis of 18,500, 14,500, and 19,000, and on December the 25th, 28,500. On December the 21st and 22nd there were considerable pain in the right shoulder and tenderness over the head of the humerus. The arm was held close to the side. Next day there was pain in the left shoulder for a few hours. On December the 28th an exploratory incision was made over the left great trochanter, but nothing was found. During the whole time since admission the fever had continued, running an irregular course, and for the three nights before the incision up to 102° F. The boy was considerably anæmic, presented hæmic murmurs over the heart, and an unduly frequent pulse, always over 100 per minute. The local signs in the left thigh had remained about the same. After the exploratory operation the boy's condition was much the same during the whole of January. The temperature was lower for a few days, and after that ranged between 98° F. and 100° F. On January the 19th and 25th the blood count

showed a leucocytosis of 22,000 and 18,000. Towards the end of the month the upper part of the thigh increased somewhat in thickness, the limitation of movement remaining the same, with pain on flexion and abduction, but none on rotation of the head of the femur. A skiagram taken on January the 27th by Mr. Greenhill showed some thickening and rarefaction of the neck of the femur and about the epiphysis of the great trochanter.

On February the 8th there was evidence of a little pus under the scar of the former operation. The wound was opened up and a probe could then be passed down to bare bone. Cultivations made from the pus yielded a growth of Staphylococcus aureus.

The boy's condition remains much the same. He is not quite as anæmic as on admission, and he appears to have gained flesh a little. His pulse rate still remains unduly high. The leucocytosis yesterday was 14,800.

The diagnosis and the treatment of this case are of great interest. In the first place the disease had to be differentiated from acute rheumatism and from hip-disease. In view of the anæmia, the leucocytosis, and the affection of the shoulders, temporary though it was, I took the view that the case was one of pyæmia, starting in or about the hip-joint. At present I am inclined to think that the actual diagnosis lies between an epiphysitis of the great trochanter, with spread of the inflammatory process to the neck of the femur, and a very subacute form of osteomyelitis starting in the lower part of the neck of the femur where it joins the diaphysis.

The prognosis is uncertain as to duration and ultimate result. In all probability further surgical treatment will be necessary, and possibly a sequestrum will form and have to be removed.

(*Exhibited February the* 17*th*, 1905.)

Discussion.

Mr. Douglas Drew said a point which arose was as to whether the pus which had appeared was the result of the

operative procedure adopted. There was much to be said in favour of the view put forward by Dr. Cautley, *i. e.* mild pyæmia, because the other joints were simultaneously affected. He had not examined the skiagraph, but from what Dr. Cautley had said there was thickening of the neck of the femur and trochanter and the upper portion of the shaft, showing there must be an extensive lesion. Also, as there was pyrexia, he thought further exploration should be carried out, to examine the condition and see what the extent of the disease was, and also to establish free drainage. He recently had a somewhat similar case, that of a child admitted with subacute symptoms about the hip-joint. It was a question whether that case should be operated upon at once. He waited two or three days and the symptoms appeared to be subsiding, but as he found there was fluctuation he cut down on the abscess, which was pointing in front, and found that it went down to the vicinity of the trochanter. There was no disease of the hip-joint, and he could not make out any disease of the trochanter. The abscess healed, and the child went out well in six weeks. He thought it must have been suppuration in one of the bursæ in the neighbourhood of the joint.

55. HEMIPLEGIC SPASTICITY AND INCO-ORDINATION IN A CHILD OF FIVE.

By EDMUND HOBHOUSE, M.D.

The first symptoms were noticed about a year ago, when a weakness of the right arm was seen. He had a convulsion in July, 1903, and was in the Sussex County Hospital for a few days just before he was brought to the Children's Hospital with pneumonia, and six months before any nervous symptoms appeared. He was re-admitted to the Children's Hospital in February, 1904, and soon after admission began to throw out his right leg, much as he does now. The symptoms seemed to improve for a time under potassium iodide, but soon relapsed again, and got steadily worse, inco-ordination and coarse tremor on attempted movement being very marked. The condition has been fairly stationary now for about eight months.

The optic discs are normal, and there are no other symptoms. The mental condition is quite good, and the child seems well in other ways. The right knee-jerk is almost impossible to obtain.

(*Exhibited February the 17th*, 1905).

Discussion.

Dr. F. Parkes Weber thought that if there were no symptoms pointing to cerebral tumour, such as optic neuritis, or convulsions, or headache, the most probable explanation suggesting itself was some form of obliteration of small arteries on one side of the brain. This might be syphilitic, but he did not think all cases in which there was obliteration of arteries affecting some part of the cerebral motor tract were necessarily syphilitic.

Dr. F. J. Poynton said he was not convinced that there was spasticity of the limb, but there was inco-ordination. It was difficult to believe it was a slow-growing tumour if there was no rigidity. He noticed there was a flexor, not an extensor, response. He had no alternative diagnosis to offer, but the condition seemed to have come on after an attack of pneumonia, and might possibly be a cerebellar lesion.

The Chairman (**Dr. Edmund Hobhouse**) (Brighton), in reply, said he should have called it a case of inco-ordination rather than of spasticity. There was some rigidity at times and at others it was not marked. The notes of the case were lost, which prevented him giving all the particulars. He was prepared with no hypothesis, but had been inclined to think it was cerebellar.

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56. A CASE OF SYPHILITIC ARTHRITIS.

By ARTHUR EDMUNDS, B.S.

The patient, a boy, aged 7 years, has had a painless swelling of the right knee for two years. Treatment has been by splinting until recently, since when mercurial treatment has been adopted. Marked diminution in swelling has followed. A skiagram shows thickening of the synovial membrane, without bony or other changes. His mother has

had five miscarriages; she was also infected with "eczema" from the patient, which produced marked scarring about the nose. (*Exhibited February the 17th*, 1905.)

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57. A CASE OF SYPHILITIC ARTHRITIS.

By G. A. SUTHERLAND, M.D.

The patient, a boy, is 12 years old. A month previously swelling of both knees had been observed, more marked in the right. He had suffered no pain except after prolonged exercise. He had never suffered from rheumatism or other acute illness, and, apart from the condition of the knees, had no subjective or objective signs of ill health. Both knee-joints were markedly swollen and contained free fluid, but there was no thickening, or tenderness, or heat, or limitation of movement, apart from that caused by the swelling. The muscles above and below the joint were slightly wasted. There was no family history obtainable, and the boy himself could not remember any previous illness. There was slight blepharitis in both eyelids, but the fundi and media were healthy. Around the alæ nasi there was some scarring. The teeth were healthy. The facial expression might be characterised as syphilitic or, at least, degenerate.

The diagnosis in this case was based on the occurrence of a painless fluid swelling of both knee-joints, unaccompanied by other manifestations of disease. The absence of pain, of any pyrexia, of other joint lesions, and of cardiac involvement was sufficient to exclude rheumatism. Osteo-arthritis and tuberculous disease could also be excluded. Dr. Sutherland asked for information as to the results of treatment in these cases. Some writers said that rapid recovery usually followed the use of mercury and iodide of potassium. His personal experience had been that these drugs did not produce any marked effect, and that the

disease ran a prolonged course, ending in spontaneous recovery. He referred to another case at present under his care: A girl, aged 19 years, had her first attack of arthritis, involving both knees, at the age of 8, her second at the age of 17, and the third a fortnight ago, when the right wrist was also involved. She had also suffered from interstitial keratitis. (*Exhibited February the 17th*, 1905.)

Discussion.

Dr. F. J. Poynton said it had been recently pointed out by Paton, and also by continental writers that there were various types of syphilitic arthritis, some of which were simple synovitis and others osteo-arthritis, and others in which there was a peri-articular swelling. The prognosis was very different in those various types. Where there was a good deal of peri-articular swelling the response to mercury seemed to be very slow. Simple synovitis quickly reacted to treatment. Syphilitic deafness, coming on at the same time as the joint changes, sometimes occurred.

The Chairman (**Dr. Edmund Hobhouse**) (Brighton) said such cases were of very great interest, and he thought the majority of them came rather under the surgeon than under the physician. He remembered when fifteen years ago the coincidence of interstitial keratitis with double effusion into the knee-joint was first pointed out by some ophthalmic surgeon, and since then he thought it had been well recognised that at the time when interstitial keratitis was first apt to appear there was also likely to be a painless effusion into the knee-joint. There seemed little doubt that Dr. Sutherland's case was one of that kind. Double painless effusion into the knee-joint was suspicious of syphilis, as there was scarcely anything else which could cause it. With regard to Mr. Edmunds' case, he supposed a single joint affection was rather rarer than double.

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58. ACQUIRED CLUB FOOT WITH MARKED HEREDITARY HISTORY.

By DOUGLAS DREW, B.S.

The patient, a female child, aged 8 years, developed pes cavus with talipes equinus varus at age of 6 years. Her

mother was similarly affected, her brother and grandmother also, and in all the deformity appeared at about the same age. The mother's aunt was also affected, and another aunt had a son who also developed club feet.

The child presented no evidence of any nervous disease, beyond that the knee-jerks were exaggerated, and the mother showed no signs of any nervous lesion. He invited physicians to say whether there was likely to be any underlying nervous lesion.

(*Exhibited February the* 17*th,* 1905.)

Discussion.

Dr. Edmund Cautley said he did not think there was any evidence of nerve-disease; he regarded it as simply a transmission of an hereditary peculiarity. The club foot had been transmitted through different generations, in the same way as superfluous fingers or toes, or even a characteristic nose.

59. EXCISION OF BOTH HIP-JOINTS FOR TUBERCULOUS DISEASE.

By DOUGLAS DREW, B.S.

The disease first appeared on the left side and was treated for some months in a double Thomas's splint, when an abscess developed. Excision was performed in July, 1905, the head of the bone being removed and the synovial membrane dissected away. It healed by first intention. Some months later a small tuberculous focus was excised from the scar; this did not extend deeply. As the result of a fall out of an invalid cart upon the right hip disease developed and an abscess formed. It was treated in the same manner as upon the left side, and healed by first intention.

It is now seven months since the second operation. The child is in good health. The joints are ankylosed in a good position, there being no tendency to adduction.

(*Exhibited February the* 17*th,* 1905.)

60. A CASE OF SPASTIC DIPLEGIA WITH OPTIC ATROPHY.

By ERIC PRITCHARD, M.D.

The case is one of spastic diplegia in a child aged 2 years. The father is stated to be consumptive and the mother has a skin lesion of the face which appears to be lupus. The mother has had seven children, of whom three, including this one, are alive; the remaining four died during early infancy. There have been no miscarriages, but one of the deceased infants was born prematurely. The labour in the case of this child was difficult and prolonged, and the presentation that of the head. No instruments were used. The present condition, as far as general nutrition is concerned, is apparently good. There are no obvious stigmata of degeneration, except that the external genitals are somewhat undeveloped and that the head is distinctly microcephalic (17½ inches in circumference). The ordinary organic functions, such as digestion, excretion, etc., appear to be quite normal and the child is clean in her habits. The spastic rigidity is almost universal, including that of the muscles of the head; it is more marked on the right side of the body. The child is quite incapable of performing any voluntary movement of the limbs. Owing to complete incoordination and spasm, she has never succeeded in grasping any object with her hands, and has made no attempts at walking. The knee-jerks are somewhat difficult to elicit owing to the spasm of the muscles, especially on the right side; on the left they appear slightly exaggerated. There is no ankle clonus, but Babinsky's reflex is well marked in the hallux of the right foot. In both arms there are almost constant choreiform movements which take the form of periodic extension and separation of the phalanges of the hand, and jerky athetoid movements of the arms. The child is subject to frequent fits, sometimes six or seven in the hour; they last only a few minutes and commence with a convulsive salaam, then a short period of tonic contraction of the extensors, and finally there is a phase of apparent unconsciousness,

during which time the child remains perfectly still, rather huddled over to the left, and in a condition of complete exhaustion. The first attack of this nature is stated to have occurred on the third day of life. The child shows no signs of intelligence; she takes no notice whatsoever. She has well-developed hyperacusis; she turns round in the direction of sounds, and sometimes has a convulsive seizure on hearing a loud noise. She appears to be completely blind, although the movements of the eyes are good in all directions, and the pupils react to light. The condition of the discs is one of atrophy; they are extremely pale, and the edges well defined. She is completely aphasic, but the sensations of taste, smell, and touch appear normal.

The diagnosis presents difficulties, although in its main essentials the case might be classified as a birth palsy; there is no clear evidence that any damage was done to the brain during the time of birth, and it is difficult to see how any hæmorrhage which could be responsible for such extensive spasticity and for the optic atrophy, could have been otherwise than fatal at the time of its occurrence.

On the other hand, if the case is to be regarded as one of congenital defect in the development of the nerve elements, the remarkably good condition of the organic functions and the absence of physical stigmata of degeneration require explanation, as also do the repeated convulsions and choreiform movements. The case certainly has similarities with amaurotic family idiocy, although no other member of the family appears to be affected in the same way, and the pathognomonic signs of this disease, namely, macular changes, are absent. On the whole I am inclined to regard the case as one of microcephalic congenital idiocy due to arrested or perverted development of the nerve elements, possibly the consequence of the tuberculous condition of the parents. (*Exhibited January the* 17*th*, 1905.)

Discussion.

The Chairman (**Dr. Edmund Hobhouse**) (Brighton) said such cases were always of considerable interest, and it was

generally difficult to explain the extent of the lesions found on any available theory, as Dr. Pritchard had pointed out. He had seen a number of such cases within the last three or four years, but there was generally extreme difficulty in examining the eyes. In two or three of his cases there was pronounced convergent strabismus, so that it was almost impossible to get a sight of the discs. In the present case it looks as if the labour had something to do with it. Many such cases had been reported in which there was considerable injury to the cerebral hemispheres during labour, but there are others, again, in which there was no evidence of instrumental injury or of any pressure during birth. He exhibited a case a few days ago in which labour had been easy, but there was extensive damage of that type; the legs were crossed, there was convergent strabismus, but the child was fairly intelligent, though the spasticity was extreme.

Dr. Edmund Cautley said Dr. Pritchard had given such a clear description that he could not agree it was congenital microcephaly. He did not think that a condition which developed apparently three days after birth could be spoken of as congenital. He inclined to the view suggested by Dr. Pritchard's description, that in all probability some hæmorrhage had taken place a few days after birth, or that some injury was done to the brain at birth, either from hæmorrhage or pressure, and that some encephalitis had been produced, setting up secondary sclerosis. Such an explanation would account for the blindness. There might be an extensive meningeal hæmorrhage over the whole surface of the brain, not necessarily very thick but widely diffused, causing encephalitis and damage to the visual centres in the ophthalmic lobes. The child was definitely microcephalic, but it seemed to him it was one of secondary development rather than a congenital condition. He had seen cases develop even later in life. One baby was born apparently perfectly healthy and remained so for several months, undergoing apparently normal development, until she suddenly started having fits and developed all the signs of encephalitis, after which she became a microcephalic idiot, but without any spasticity. In that case the evidence was in favour of an encephalitis affecting the frontal lobes.

Dr. G. A. Sutherland remarked that the unusual feature in the case was the optic atrophy, of which no very clear explanation had been forthcoming. He suggested that one other possible cause was syphilis. When there was optic atrophy in such cases it was suggestive of a syphilitic origin. He did not know whether there were any changes in the choroid, but he could recall cases in which optic atrophy had been associated with syphilis. He was getting more sceptical about the theory of meningeal hæmorrhages at birth. If a hæmorrhage occurred sufficiently extensive to produce the changes seen, he thought it

would have been a fatal one. The other theories which had been advanced, viz. direct injury to the brain and the non-development of that organ, were more plausible.

Dr. Eric Pritchard, in reply, said he thought Dr. Cautley misunderstood him. He (Dr. Pritchard) concluded it was a congenital condition. Although the first symptom was only noticed on the third day, it was possible the child had a fit before that. He did not suppose that at the third day a hæmorrhage took place which could account for the subsequent symptoms. He agreed with Dr. Sutherland that if a hæmorrhage had occurred sufficiently large to cause the symptoms now seen, the result must have been death.

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61. CONGENITAL ABSENCE OF GALL-BLADDER AND BILE-DUCTS.

By EDMUND HOBHOUSE, M.D.

The specimen was removed from a female child, aged 7 months, jaundiced from birth. The urine was somewhat milky, but contained no bile salts or pigment, and no albumin. The fæces were white. The child took food well, and was not sick, but was much emaciated, weighing only 9 pounds at the time of its death, at 8 months. The liver weighed 8 ounces; it showed no trace of gall-bladder or bile-ducts. Microscopically there was well-marked cirrhosis, the changes being interlobular, and in places intra-lobular. Bile secretion had evidently gone on actively.

(*Exhibited February the 17th,* 1905.)

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62. INTERSTITIAL NEPHRITIS IN A CHILD OF SIX AND A HALF WITH RENAL RETINITIS.

By EDMUND HOBHOUSE, M.D.

The chief points of interest in this case from which the kidneys exhibited were taken were briefly that the early symptoms rather suggested cerebral than renal

mischief; the fits resembled those due to a pontine lesion; that the hæmorrhage seemed to be a relief to the kidneys, the flow of urine increasing during its continuance; and that the ocular symptoms were severe. In other respects the case resembles others of the same kind. The cause was probably scarlet fever. There was no evidence of congenital syphilis, nor was there anything to explain the rapid course of the disease. The only point in the family history was the presence of tuberculosis on both sides.

B. B—, aged 6½ years. At 3 years of age she had sore-throat, which her mother believes was scarlet fever. This was followed by some swelling of the feet and legs; otherwise she was healthy till four months ago, though not strong. Then she was sick in school, and has suffered since from headache and sickness occasionally. On November the 23rd, 1904, at 3 a.m., she had general convulsions, and was admitted into the hospital the same day.

State on admission.—A delicate, refined-looking child, thin but fairly well developed, somewhat collapsed, and dull and drowsy. Two hours later she was very sick, and remained so for some hours, quite irrespective of food. Quite conscious, but does not speak; no paralysis or rigidity, no headache; abdomen very retracted. On November the 25th, at 8.30 p.m., she was suddenly seized with convulsions; the spasms were limited to the right arm at first for about fifteen minutes, then after ten minutes' remission the right arm and left face began twitching, and shortly after that the right leg. This lasted intermittently till 4 a.m., with somewhat stertorous breathing. Chloral and bromide of potassium were given by the rectum. Urine, sp. gr. 1030, alkaline, with small amount of albumin; no casts. On November the 27th the eyes were examined, and a suspicion of renal retinitis noted. On December the 3rd the eyes were re-examined, and some soft white patches seen in the fundus; no hæmorrhages. General condition about the same, quite conscious but dull; no more vomiting. On December the 20th blood was noticed in the urine, which was not smoky but port-wine colour; no casts could be

found. The urine, which had varied from 25 to 30 ounces *per diem*, rose to between 40 and 50 ounces, and remained at that level till January the 6th, when it gradually sank till death, which occurred on January the 20th. The blood persisted till January the 10th and then disappeared. On January the 8th vomiting began, which persisted till death. No casts were discovered till the urine was centrifugalised, when a small number of hyaline casts were found, with a moderate number of erythrocytes and leucocytes. The renal retinitis on January the 10th was of the most typical character. In both eyes there were numerous hæmorrhages, large white patches, and numerous small, glistening, white dots, very extensive areas of both fundi being involved. The discs were moderately swollen. Owing to the child's mental drowsiness it was not thought worth while to test the vision. She became gradually exhausted, and died on January the 20th of simple asthenia, just about two months from her admission into hospital and the date when the renal retinitis was commencing. She had no return of convulsions or coma.

Post-mortem examination.—There was practically no sign of disease in the body except in the kidneys, of which the left was exceedingly small, weighing about half an ounce; the right weighed $2\frac{3}{4}$ ounces and was a good deal puckered. Dr. Bushnell, pathologist to the Sussex County Hospital, who kindly examined them, reports that they are probably secondary to a mottled kidney caused by a former acute or subacute nephritis. The brunt of the lesion has fallen on the glomeruli, which are the seat of proliferation of the epithelium, of capsulitis and a hyaline change. The arterioles are thickened. There are no gummata; the naked eye and histological characters favour a secondary rather than a primary interstitial nephritis.

(*Exhibited February the* 17*th*, 1905.)

63. A CASE OF ALOPECIA UNIVERSALIS.

By OTTO GRÜNBAUM, M.D.

H. J—, a male, aged 9 years. Two years ago the hair on his head came out in patches, these patches coalesced, and complete baldness occurred within a year of the beginning of the disease. As soon as the baldness was complete the eyebrows and most of the lashes seemed to fade away. The nails are normal. No history of disease or worry.

(*Exhibited March the 17th*, 1905.)

Discussion.

Mr. George Pernet remarked that those cases of alopecia areata terminating in a generalised alopecia were not very common, but in skin departments one naturally saw more of them than in a general medical department. There were at present two or three cases in children attending the University College Hospital, and he had seen several instances of it altogether in the last ten years. As Dr. Grünbaum had found, they were very rebellious to treatment, and very discouraging to both patient and doctor. In some cases he had succeeded in getting a growth of hair to appear, but, after continuing there for a few weeks, the whole of it had come off, and it became necessary to start the treatment afresh. It was necessary to persevere, and in the present case the age was in favour of the patient. One case attending University College Hospital had gone on for many years getting crops of hair and losing them again. It was difficult to label any germ as a specific cause of the condition. There was also the neurotic view to be taken into account. It was very necessary to keep the child's health at the best possible level, because he had found that if malt extract and hypophosphites were given to badly nourished children that helped the treatment very much. Thyroid treatment had not been of much use. Stimulating lotions should be employed, the formulæ for which were endless. Kromayer had advocated light treatment, but the speaker did not think that was of more use than a stimulant local treatment; indeed, that was how the light acted.

Dr. C. W. Chapman called attention to the fact that there were patches of hair on the hands of the patient.

Dr. Otto Grünbaum, in reply, remarked that it was not strictly a case of alopecia universalis, because there were patches of hair on the hands and a few eyelashes on the lower lid of the left eye.

64. A CASE OF LATE RICKETS.

By ROBERT HUTCHISON, M.D.

The patient, a boy, aged 9 years and 8 months, was brought to hospital suffering from bending of the legs, which had been getting rapidly worse for some months, and for a few weeks had incapacitated him from walking. The history was that he had been delicate since an attack of diarrhœa and vomiting at 16 months. At the age of 3 years he had rickets, from which he had never completely recovered, but the condition had become aggravated recently, as stated above. He had never walked well.

He was breast-fed till 8 months old, and after that had pure cow's milk. The diet subsequently seemed to have been quite a fair one. He had had no important illnesses. He was the second of five children, of whom one, a girl, aged 8 years, is suffering from marked genu valgum and enlargement of the epiphyses of the long bones; another, aged 6 years, has slight knock-knee; and another, aged $2\frac{1}{2}$ years, has rickets and bow legs. The patient himself was fairly well nourished, but exhibited very marked enlargement of the lower ends of the radius and ulna and of the epiphyses of the knee-joint and ankles, along with typical rickety curvature of the femora and tibiæ. The skull and viscera showed no change, nor was there any beading of the ribs or deformity of the chest.

(*Exhibited March the 17th*, 1905.)

Discussion.

Dr. Leonard Guthrie said he had grave doubts of the existence of late rickets. He had never seen such a case and was rather inclined to follow the second idea suggested by Dr. Hutchison, viz. that it was a case of continuous rickets with exacerbations, which he supposed did occasionally happen in rickets. If there were skiagrams of the case, they would be useful as throwing light on whether there was a true enlargement of the epiphyses as in rickets, or whether the diaphyses were affected as in fœtal rickets or achondroplasia. He did not think

it was achondroplasia, neither did he regard it as ordinary rickets.

Mr. Jackson Clarke said that persistent rickets in orthopædic cliniques was common up to 8, 10, 13, and 14 years of age. In the cases of knock-knee and flat foot which arose in adolescents one could generally trace slight rickets which had lasted from infancy. He had three or four cases under his care in which the bone lesions were very pronounced, with thickening of the epiphyses in girls and youths of 13 and 14 years of age.

Dr. G. A. Sutherland thought that persistent rickets was not common in the experience of most medical men. He had been somewhat sceptical about that condition for some years. Considering the number of cases of rickets which passed through the hands of a physician to a children's hospital, one would expect to see it more frequently than was at present the case, especially such a striking condition as in Dr. Hutchison's patient. The history in the case was somewhat vague, as Dr. Hutchison could not tell exactly what the progress of the case had been, but the boy said he had never been able to walk at all well and he had been worse lately; therefore one might conclude that the illness, whatever it was, had never passed off entirely. The condition of the limbs was striking, because even in rickets such an enlargement of the epiphyses would be extremely unusual, and allowing for the patient's age, 9 years, the enlargement seemed to be enormous. But perhaps that was intensified by the fact that there was considerable muscular wasting about the joints. He asked whether Dr. Hutchison thought there might be some disease other than rickets present. If it were rickets, it was curious that the other signs of the disease had subsided. There was no alteration in the shape of the skull to suggest it, and no beading of the ribs. There were no convulsions or special sweating, although certainly the boy seemed somewhat clammy. He also would have liked to see a skiagram of the case, as he thought there must be something more than rickets present, though he could not give any suggestion as to what it was. He felt sure the condition was not common in juvenile life.

Dr. F. Parkes Weber said he had been converted, if he needed conversion, in recognising existence of "late" or "persistent" or "delayed" or "recrudescent" rickets by another case shown to him elsewhere, very well marked and in a much older child (aged 16 years). He also mentioned that quite recently, when visiting the British Museum, he was shown the skull of a young woman which had been found in Egypt by Mr. Hall, who was assistant in the Egyptian Department of the Museum; this skull showed a certain amount of quite symmetrical bossing over both parietal bones, of the peculiar fairly smooth but porous character which was found in disappearing

Parrot's nodes. If one could picture to oneself pumice-stone which had been worn smooth on the surface, that would represent the appearance of the parietal bosses; that is to say, although the surface was smooth it was full of tiny holes. At the margins of the patches the change from the porous to the normal cranial surface was quite gradual. He was told there was a somewhat similar skull in the museum of the Royal College of Surgeons (the specimen dated from 1843), that of a boy, aged 19 years, who died of pulmonary tuberculosis. Dr. Weber could not affirm that these two skulls were certainly from rachitic individuals, but he alluded to them because he thought late rickets had a very wide bearing and one should always be alive to it, not only in the kind of case brought forward by Dr. Hutchison, but in examining specimens which one saw by chance. He did not know what was the latest age at which bone changes occurred which could be ascribed to rickets, but perhaps some information would be forthcoming on the point.

Mr. Lockhart Mummery said he believed in late rickets, but thought the condition extremely rare. It would be very interesting if Mr. Jackson Clarke would show some of the cases to which he referred, because it had always to be considered in London that cases often went to special hospitals which were not seen in ordinary work, so that the same disease might occur in a different aspect at a special hospital, a fact which was apt to be lost sight of.

Dr. Robert Hutchison, in reply, regretted that the skiagrams had not been delivered in time to enable him to show them. He agreed with Dr. Sutherland that Mr. Jackson Clarke would not have taken the view he did if he had seen the case. He thought no one would regard the case as common. It differed entirely from mere persistent rickets because the boy had got worse in the last few years, both with regard to the curvature and the thickening of the bones. Still, he could not suggest what other disease it could be. It seemed to be more like rickets than anything else, and too much importance should not be attached to the absence of other signs. It was well known that sometimes in rickets the visceral symptoms were entirely absent and the osseous present; at other times the osseous symptoms were absent and the visceral ones present. In others the cranial deformity might be well marked and the other bones normal. Therefore the case should not be put aside merely because the skull happened to be normal. He agreed that if Mr. Jackson Clarke would bring some of the cases which he described before the Society, he would be doing its members a service. He would be much surprised if Mr. Clarke could bring forward many cases at all resembling the present one.

65. A CASE OF TUBERCULOSIS OF THE LACRYMAL SAC.

By SYDNEY STEPHENSON, C.M.

Mr. Sydney Stephenson showed a boy, aged 9 years, with tuberculosis of the lacrymal sac. Tubercle bacilli were found in scrapings from the interior of the diseased sac. There was a family history of tuberculosis and a past history of enlarged cervical glands, which had been operated on. Chronic ulceration of the nasal chambers, probably tuberculous, was still present.

(*Exhibited March the* 17*th*, 1905.)

66. A CASE OF KERATOMALACIA.

By SYDNEY STEPHENSON, C.M.

The case is a typical one of keratomalacia—that is to say, sloughing of the corneæ in a young child suffering from marasmus, symptomatic or idiopathic. The condition is rare in England. It corresponds with "Hikan," a disease that appears epidemically in Japan during the three so-called "diarrhœa months" July to September, to the South American ophthalmia Braziliana, and to the destructive keratitis common in Russia during the great Lenten fast.

The facts of the case are as follows:

Violet H—, aged 8 months, attended the Ophthalmic Department of the North-Eastern Hospital for Children on March the 16th, 1905.

History.—The baby was born at term, and then appeared to be healthy. She has been fed upon cow's milk from birth. When 2 months old she began to fall away and to

suffer from vomiting and diarrhœa. She attended the hospital as an out-patient under Dr. George Carpenter from September the 15th to October the 6th, 1904, and recovered under treatment with calomel, bismuth, and salol. Barley-water was prescribed with the cow's milk. About a month ago the baby again began to waste, and has continued to waste ever since. There has been diarrhœa (four to eight motions a day) but no vomiting. On March the 11th—that is, five days ago—"something white" was noticed over the eyes by the mother. Since March the 12th the baby has lain with her eyes covered by her hands, and has moaned incessantly. First child.

Present condition.—A miserable-looking baby, weighing (nude) 9 lbs. 8 oz. It moans and cries feebly during the whole time it is being examined. No redness or swelling of the eyelids, which are kept constantly shut. There is a slight running of water from the palpebral fissure, but no discharge of muco-pus. The eyeballs manifest little redness. Both corneæ are converted into greyish-white sloughs, except for a narrow peripheral portion, 1·5 mm. to 2 mm. wide. The limbus is slightly raised, and forms a narrow, glistening band encircling the gangrenous corneæ. There is a small triangular area of dry and greasy-looking xerosis in the ocular conjunctiva on the outer side of each cornea. At the upper and inner part of the right cornea is a small black spot, representing a prolapse of the iris at an early stage.

(*Exhibited March the 17th*, 1905.)

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67. A CASE OF TUBERCULOSIS OF THE LUNGS, THE CERVICAL GLANDS, AND THE EARS.

By G. F. HETT.

(Introduced.)

The girl is 12 years old. Three months ago "lumps" were noticed in the left side of the neck, which have in-

creased in size. The child has been dull and apathetic for a year or more. There has been a discharge from the left ear on and off for two years. "Winter cough" has occurred every year for ten years.

Her father has suffered from abscesses in the neck and has a chronic cough. Her mother died at 35 years old, of consumption.

On examination the child has enlarged glands—the largest about the size of a hazel-nut—in the anterior, posterior, and occipital triangles on the left side of the neck. They are discrete, fairly soft, and are not attached to the skin. There are also palpable glands in the anterior triangle on the right side of the neck. Glands can be felt in both axillæ and in both groins. The glands became smaller under treatment with arsenic.

Physical signs in chest.—The right apex is dull to percussion, and the note is impaired all down the right side of the chest. There is bronchial breathing at the right apex and fine and medium-sized crepitations over the upper four spaces in front and behind. She was admitted into hospital. Her temperature went up to 100° F. at night for the first three days in hospital, but the moist sounds disappeared and the temperature returned to normal after nine days. She was discharged on the tenth day.

Mr. Hunter Tod examined the ears and found chronic otitis on both sides. He thought the left might be tuberculous.

The case seems of interest especially from the point of view of the glands. The strong family history of consumption, the fact of co-existing phthisis, and the possible tuberculous disease of the ears, together with the enlarged glands, at any rate suggest that these latter might be tuberculous, especially as there is often difficulty in distinguishing certain types of tuberculous adenitis clinically from lymphadenoma.

On the other hand, lymphadenomatous glands and phthisis do co-exist, and a mixed type of gland is met with

which is partly lymphadenomatous, partly tuberculous, and, according to some observers, each condition rather predisposes to than negatives the other.

In favour of the glands being lymphadenomatous and not tuberculous in this case are the following facts:

(1) The glands are discrete, movable, and not inflamed or tender, and none fluctuate.

(2) The large number of groups of glands involved.

(3) The glands became smaller under treatment with arsenic.

The glands would fall in well with the soft form of lymphadenoma.

The blood-count shows no great abnormality, *i. e. Red corpuscles*, 4,812,000 per c.mm.; *hæmoglobin*, 75 per cent.; *colour index*, ·08; leucocytes, 12,200 per c.mm.; polynuclears, 62·4 per cent.; lymphocytes, 27·6 per cent.; large lymphocytes, 8·4 per cent.; eosinophiles, 1·6 per cent.

(*Exhibited March the* 17*th*, 1905.)

Discussion.

Mr. Herbert L. Carre-Smith regarded the case as very interesting, not only because of the affection of the glands, but also because it was a case of undoubted phthisis, and phthisis was very uncommon in young children.

Dr. G. A. Sutherland regarded the case as very interesting from several points of view. No doubt the diagnosis would be made differently by physicians and by surgeons; the latter would probably diagnose it as lymphadenoma, while the physicians would say it was tuberculosis. He did not regard the glands as lymphadenomatous. He thought that in the course of time, if the case were left alone, there would be rapid softening. He would like to hear from the surgeons present whether, knowing the child had pulmonary phthisis and chronic tuberculous disease of both ears, they would propose to excise the glands. That seemed to him to be a very practical question. Last year he saw a lady from the North of England who had advanced and advancing phthisis, with very rapid enlargement of glands in the neck. The surgeon had definitely stated that the glands should be removed at once. He believed that was done, but he had not heard. He (Dr. Sutherland) would have taken a different view, and would have counselled leaving them alone. But in the present case the matter was different: the

phthisis was chronic, and the tuberculous disease of the ear was also chronic, and the glands appeared to be going on to suppuration.

Dr. J. M. Fortescue-Brickdale (Bristol) said the last similar case he had he made a post mortem upon, was that of a child, aged 8½ years, with enlarged glands. A later symptom in that case was otorrhœa. She had two or three operations for the glands in the neck, and finally a very extensive one. She was found post mortem to have had some old pleural adhesions and enlarged tuberculous glands in the thorax, abdomen, and inguinal region. He did not think this was a favourable case for surgical interference, nor was any case where there were multiple tuberculous foci. In the case alluded to the temperature rose immediately after the last operation, which appeared to have been the starting-point of the acute miliary infection which proved fatal.

Dr. Leonard Guthrie thought the incidence of pulmonary phthisis in children was not by any means uncommon, but most frequently it was associated with tuberculous glands, and those glands were for the most part situated in the thorax. How they became infected was not very clear. Professor Sims Woodhead said it was directly through the tonsils, and that the glands through the neck and mediastinum became affected from the primary affection of the tonsil. He (Dr. Guthrie) was doubtful about that, as he thought the glands in the thorax might be primarily affected. Still, it was not clear to him how it occurred, unless it was through the air. He asked whether any examination of the chest had indicated enlarged mediastinal glands.

Mr. G. F. Hett (introduced), in reply, said that there was no sputum, and that Mr. Hunter Tod had said that although the ear condition was probably tuberculous, he could not diagnose it without a bacteriological examination, and perhaps not then, because even in tuberculous otitis the bacilli were few and might not be found. Dr. Sutherland would be better qualified to give an opinion about the mediastinal glands, as the case was under his charge in the ward, but he believed that they were not enlarged.

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68. A CASE OF TREMOR.

By J. CHARLTON BRISCOE, M.B.

The patient, a girl, aged 13 years, was well developed. She was in the fifth standard, intelligent, but rather inclined

to worry over her work. The tremor affected the right arm alone, was very rapid and better felt than seen. It was constant when awake, but absent during sleep. It was accentuated after prolonged exertion and diminished but did not disappear with rest in bed. When the arm was hanging by the side the tremor was least marked. In this position the muscles affected were the infra-spinatus and posterior portion of the deltoid. With the arm extended, in addition to the afore-mentioned muscles, the pectorales major and minor, the serratus, the rhomboids, trapezius, levator anguli scapulæ, the whole of the deltoid, and sometimes the triceps showed a tremor. The muscles of the forearm and hand were not involved. There was no muscular wasting or hypertrophy, and the electrical reactions were normal. Movements were correctly performed, except that occasionally water was spilt when carrying a cup to her mouth. The shoulder-joint moved freely and did not grate. The nervous system otherwise was normal. The fundus oculi showed no pathological changes, and there was no alteration of sensation. The thoracic and abdominal viscera and the urine were normal. The past history was uneventful. The condition had lasted three months. The onset was sudden, with slight anorexia and lassitude. There was a history of insanity in one uncle, and a sister had apparently died in a fit.

(*Exhibited March the* 17*th*, 1905.)

Discussion.

Dr. Leonard Guthrie asked whether at the onset of the illness there was any pyrexia. He understood there was slight malaise, but nothing more specific. It suggested to him a mild encephalitis, and it might be due to thrombosis of some of the finer vessels of the left cortex; he did not think it a case of disseminated sclerosis. The movements of the right arm were certainly in favour of that view. The tremors were not those of chorea, and they did not seem to suggest a functional element. They were somewhat increased in voluntary movement, which was also in favour of a slight lesion on the opposite side of the brain. There was no doubt about nystagmus being present that afternoon, and it was more marked when the eyes deviated to the right side; it occurred only on extreme deviation of the eyes.

He would not attach very much importance to that. It would be very young for disseminated sclerosis. Of course, the term "functional" could be applied to anything which was not understood, but he believed that in "functional" cases at that age there was a pathological substratum to account for the symptoms. There were a considerable number of cases on record which pointed to slight encephalitis as the cause of tremors.

Dr. G. A. Sutherland thought if one took into consideration the age of the child and the nervous history, it might be explained by the indefinite term "functional tremor." The nystagmus also was of indefinite type, and he could not convince himself that there were definite nystagmoid movements present. A little mental and moral treatment might well be tried, including strong applications of the faradic current.

Mr. Sydney Stephenson said there seemed to be a difference of opinion as to the presence or the absence of nystagmus. Dr. Briscoe said he examined the fundi and they were steady, and that was conclusive evidence that at the time of that examination there was no nystagmus; for if there was one particular test for nystagmus more delicate than another, it was to attempt to examine the fundus of such a case. He (Mr. Stephenson) had looked at the eyes, and nystagmus in the ordinary sense was not present, but in the somewhat imperfect light he thought there were some nystagmoid jerks when the eyes reached their end-positions. It would be desirable to know whether nystagmus was present or not, because it had a bearing on the diagnosis, particularly if insular sclerosis was a possible diagnosis. Fifty per cent. of the patients with that disease had nystagmus or nystagmoid movements. He was inclined to differ from the view that insular sclerosis was uncommon in children of that age. Kempherstein * had published an elaborate analysis of thirty-seven cases of insular sclerosis, and in 18 per cent. of them the disease occurred between the ages of 10 and 15 years.

Dr. J. C. Briscoe, in reply, said he had excluded encephalitis because there had been no alteration in the condition during the last six weeks, no alteration in the knee-jerks or in the face, and there was the definite tremor in the muscles mentioned. There was apparently nothing remarkable about the onset, never any unconsciousness, but the history given by the mother was not very clear. He was inclined to Dr. Sutherland's view that the case was functional, and agreed that the battery would help the cure. He had not seen any cases of disseminated sclerosis as young as the present patient, but he thought they were not very uncommon about the age of 15.

Postscript.—The tremor subsided entirely and somewhat suddenly about fourteen days after the case was shown.

* Kempherstein, 'Archiv für Augenheilkunde,' 1903, Bd. 49, p. 41.

69. JAUNDICE IN A CHILD, WITH CHRONIC ENLARGEMENT OF THE SPLEEN.

By F. PARKES WEBER, M.D.

Eliza K—, aged 12 years, a rather delicately-built girl, is moderately jaundiced. The fæces are sometimes well-coloured, sometimes pale. The urine, which is free from albumin and sugar, gives at present no distinct Gmelin's reaction for bile-pigments, but recently gave a slight one. The spleen can be felt extending three finger-breadths below the costal margin in the left nipple line, and the area of splenic dulness is found to be greatly enlarged by percussion; it measures $3\frac{3}{4}$ inches in breadth, and $6\frac{1}{2}$ inches in length. The liver cannot be felt, and is apparently not enlarged. The other abdominal organs and the thoracic organs show nothing abnormal, excepting that there is (at least, at times) a slight systolic murmur to be heard over the pulmonary area of the heart. Pulse, about 84 per minute, very much increased in frequency on excitement. The teeth are not very well formed, but are not distinctly "Hutchinsonian." Ophthalmoscopic examination shows nothing abnormal. A blood-count in February gave: Red cells, 4,824,000 in the cubic millimetre; white cells, 5,100. Following is the present differential count of the white cells: Polymorphonuclears, 65·2 per cent.; lymphocytes, 30·0 per cent.; large mononuclears, 4·8 per cent. The lymphatic glands are not enlarged. There is no fever, and the child seems cheerful and complains of no pain or abnormal tenderness anywhere. The present attack of jaundice apparently commenced about the middle of February, after the child had complained of pain in the left (*not right*) side. According to the mother, about that time she vomited twice. Six years ago she had two attacks of jaundice, lasting two months and one month respectively, separated by an interval of three months during which she was free from jaundice. Though the

child presents no obvious signs of congenital syphilis, the history in regard to the mother's other children is somewhat suggestive. The mother has had eleven children altogether, out of which six were born dead and two died early. There has not been much change in the patient's condition in the last three weeks, during which she has been under observation at the German Hospital. Two possibilities suggest themselves. Firstly, the splenic enlargement and the attacks of jaundice may be connected with congenital syphilis. Secondly, although there is no family history of jaundice in the present case, the condition may be allied to certain recorded "family" cases of more or less chronic jaundice (or recurrent attacks of jaundice), associated with chronic splenomegaly. A family history of such cases was described by Dr. Claude Wilson ('Clin. Soc. Trans.,' vol. xxiii, p. 162, and vol. xxvi, p. 163). A family series of similar cases has been published by Dr. J. A. Arkwright ('Edinburgh Medical Journal,' January, 1903, p. 52), consisting of father, one son, and one daughter, in a non-malarial country, with considerably enlarged and hardened spleens, persistent slight jaundice, and occasional attacks of fever, vomiting, and temporary increase of the jaundice. In both a mother and her son, shown by Sir Thomas Barlow and Dr. H. Batty Shaw at the Clinical Society of London (May the 23rd, 1902), there was splenic enlargement and a slight degree of persistent jaundice, but the mother, owing to the enlargement of the liver, was perhaps an instance of actual biliary cirrhosis. Among the cases of "ictère chronique splénomégalique" described by Lereboullet ('Les Cirrhoses biliaires,' Paris, 1902, pp. 447–464), there are some family groups probably comparable to these.

(*Exhibited March the 17th,* 1905.)

Discussion.

Dr. G. A. Sutherland said it was difficult to give a diagnosis without careful examination, but it might belong to the rare class of case mentioned or might be syphilitic. He agreed it was worth while to try the effect of antisyphilitic treatment, but suggested a combination of mercury with the iodide, as it was the

mercury which was the effective drug in such a case. He noticed the spleen rose very high in the axillary region, and he would like to know whether there was any suggested reason for that enlargement upwards. Did Dr. Weber recognise some conditions in which the spleen enlarged upwards and others in which it enlarged downwards? He hoped there would be an opportunity of seeing the case again.

Dr. Parkes Weber, in reply, in regard to enlargement of the spleen, said he thought that whether it took place upwards or downwards, and whether it projected below the costal margin in front or at the side, depended very much upon its attachment, which varied congenitally very much in different individuals.

Postscript.—The patient left the hospital on July the 2nd, 1905. At that time she was free from jaundice, with the exception of a very faint yellow tinge in the sclerotics, and her general health appeared to be good. The spleen, however, had not at all diminished in size in spite of treatment, firstly by a course of mercuric inunction with the internal use of potassium iodide, secondly by a course of exposure of the splenic region of the body to Roentgen rays. The blood-count on July the 1st gave 4,696,000 red corpuscles, and 3400 white corpuscles to the cubic millimetre of blood. The edge of the liver could just be felt below the ribs. The urine was free from albumin.

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70. NOTES OF A CASE OF HYSTERIA IN A GIRL TEN AND A HALF YEARS OF AGE.

By CHARLES W. CHAPMAN, M.D.

The patient, a girl, aged 10½ years, with whose family I was well acquainted, was brought to me in December, 1884, in consequence of her having failed in health, and because she had little or no desire for food. She was the youngest of a family of five, the other four having always had very good health. The patient had never been robust, but had not suffered with any serious illness. Both parents

were healthy, the father very intellectual, and the mother of average health, though slightly neurotic. The child had been petted a great deal and allowed to have her own way and the parents considered her to be delicate. Careful examination failed to discover any organic disease. The appetite was bad and the tongue dirty. It transpired that the child had been allowed to have food whenever she wished; moreover she was consulted as to what kind of food she preferred. I advised she should have her meals with the other children, and that no difference be made in her food from that given to the brothers and sisters. I prescribed Hydrargyrum c̄ cretâ twice a week, and a rhubarb with soda mixture thrice daily before food. She was to have plenty of outdoor exercise whether she was agreeable to it or not. Finally, she was to be seen at intervals by the family doctor.

The reports I received from time to time were by no means satisfactory. The patient had become morbidly affectionate to her parents, and she would not allow herself to be left without one or the other of them being with her. I was informed my suggestions had only partially been carried out. The doctor had advised change of air and scene, and the child was taken to Brighton. Later on she went with the family to Broadstairs. While at the latter place listlessness gradually replaced the former excitability, so much so that she would scarcely answer an ordinary question. In February, 1885, I saw the patient in consultation with Mr. Cockell, under whose care she had been all along. Again there was no evidence of organic disease. I repeated my advice as to moral and other treatment with increased emphasis, and gave a good prognosis provided the instructions were carried out. Later on I was informed that as the child had not improved the parents had taken her to two well-known physicians, one of whom was an eminent neurologist. The opinion of these gentlemen was that the child was in all probability suffering from cerebral tumour, and they gave a grave prognosis. One of these consultants was so sure of his diagnosis as to mildly

suggest a post-mortem examination when the fatal event happened, and expressed a wish that he might be present at the autopsy. As time went on the patient became weaker and the symptoms increased in severity. A second neurologist was called in, for by this time the child was bedridden from complete loss of power and sensation in the legs. On this occasion no definite opinion was given. By October the patient became emaciated, she passed both urine and fæces under her, and she declared she could see but little, and was at times quite blind. As the parents were worn out with anxiety and watching, I was again asked to see the child, with the assurance that whatever I ordered would be strictly carried out. It was a singular thing that, although the child was supposed to be nearly blind, she was constantly asking for flowers, and expressed satisfaction whenever a fresh plant was brought. On my arrival I found the bedroom crowded with pot plants and cut flowers. I was told that for the last two weeks the child had had a dreadful screaming fit every afternoon at about 4.30, so loud and piercing as to attract passers-by. The patient strongly resented any examination at first, but I succeeded subsequently in doing all I wanted to do. The blindness was proved to be non-existent, and an opthalmoscopic examination showed the discs to be perfectly healthy; in the end I was strengthened in my opinion that the case was functional rather than organic. I advised complete separation of the patient from her friends and relations, and such other measures as would be necessary in a case such as this one was believed to be. As the finances of the parents had been severely strained over the illness, arrangements were made for the patient's admission into Great Ormond Street under Dr., now Sir Thomas, Barlow, in October. The progress of the case after admission can be summed up in a few words. The screaming did not occur once after leaving home, and by the end of a month the patient was able to walk. Later on the child left the hospital for a lengthened stay in the country, where she soon regained normal health. During the interval of twenty

years the patient has enjoyed very good health, and has had no return of the old symptoms.

The diagnosis in cases of the character of the one now related is not infrequently beset with difficulties. The exclusion of organic disease may require repeated examinations to justify a positive opinion, as this case illustrates. It is extremely unlikely that the two physicians who were consulted would have been misled, as they certainly were, had they had the opportunity of seeing the patient a second time. I had the advantage of knowing the family intimately, and of having seen the patient at intervals during her whole life.

The importance of a correct diagnosis in cases like the present cannot be overrated, for a correct prognosis and success in treatment depend entirely upon it. I make no apology for bringing this case before the Society, for I am convinced from my own experience that not a few saintly sufferers who are at once the objects of the admiration and sympathy of their friends would have made useful members of society had the neurotic bases of their illnesses been early recognised and appropriate treatment adopted.

It is necessary in these cases to pay great attention to the digestive organs, for derangement of these will initiate an hysterical condition in patients predisposed to it. This point is insisted upon by Trousseau, in Vol. IV of his Clinical Lectures published by the Sydenham Society. The author states: "Effects of dyspepsia on the nervous system are analogous to those from which hysterical women suffer, such as analgesia and partial anæsthesia." In the present instance dyspepsia and constipation were more or less in evidence during the entire illness, and it is to be noted that the original suggestion of an occasional dose of calomel and regular doses of rhubarb with bicarbonate of sodium was followed after the patient went to the hospital.

The late Dr. West's remarks on cases of the character of the one just described are singularly appropriate. Among the causes he gives "vanity and indolence, also a sense of importance in finding in the household everything arranged with reference to the patient's condition. This

morbid craving for sympathy and love are not infrequently indulged and gratified and exaggerated by the conduct of a foolishly fond mother. Separation of the child from the latter is the most effectual mode of treatment."

The prognosis in hysteria in children is generally good, but if there are cataleptic symptoms epilepsy may be feared.

(*Read April the 14th*, 1905.)

Discussion.

The Chairman (**Mr. R. Clement Lucas**) remarked that the case was a very interesting one of extreme hysterical or functional disturbance in a child. No doubt many of those present had also met with such cases, though probably not so severe. A class of case in children which came under the surgeon in that connection was in those suffering from enuresis. Nothing but a little moral control was necessary in order to cure the condition.

Dr. G. E. Shuttleworth (Richmond) said one occasionally met with children of neurotic parents who evinced certain peculiarities about their food which in an adult would be described as hysterical. That, he believed, was frequently owing to the bad management of the parents and the indulgent manner in which such children had been brought up. Not infrequently such children were brought to him with a list of what they could and could not eat. The mother would often say that the child could not eat vegetables and fruit, or either, and if those aliments were given the child would be sure to be ill. To give such a child oatmeal porridge was considered to be most heinous. But when such children were introduced to the general *régime* of the establishment with which he was connected, in a few weeks they would be taking the ordinary miscellaneous dietary prescribed for the other children, and there was no further difficulty. He believed the occurrence of hysterical symptoms in neurotic children was more common than English writers would lead one to suspect. French writers attached much more importance to it than English, and Trousseau and Briquet gave examples of such cases, with anæsthesia and analgesia, in quite young children. The thanks of the Society were due to Dr. Chapman for narrating the case with such interesting detail.

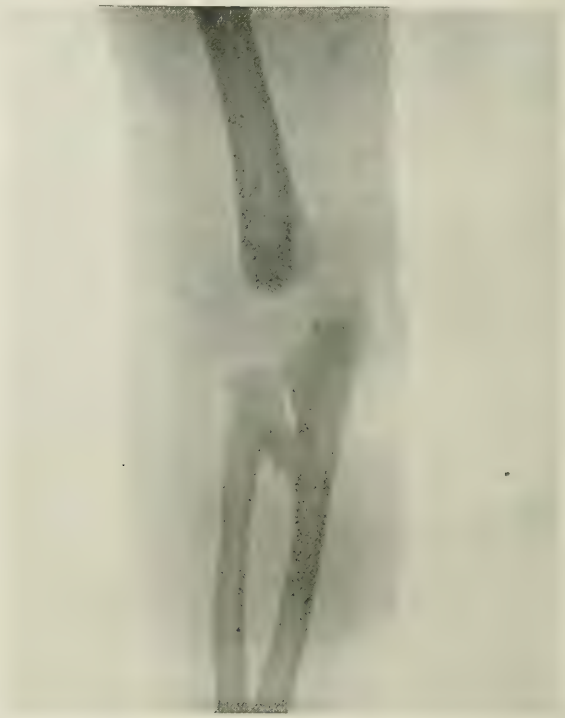
Dr. C. W. Chapman, in reply, remarked that in the present days of elaborate dietaries there was a danger of people and children becoming abject slaves to the list which was drawn up for them, so that their stomachs were unfit for ordinary food, and they would not take it; in fact, the stomach became as obstinate as the patients themselves. By putting them on ordinary food they in most cases got much better.

71. A CASE OF CONGENITAL DEFORMITY OF BOTH FOREARMS.

By R. C. DUN, C.M.

John W—, aged 5 months, was seen at the Liverpool Infirmary for Children. Both forearms and hands were firmly fixed in the position of full pronation, and manipula-

Fig. 12.



tions failed to produce even the slightest degree of supination. On examination the bony points about the elbow and wrist were found to occupy their normal positions. The range of movement at the elbow and wrist joints was normal. The deformity had been noticed a few days after birth. The labour had been easy, and the child was otherwise well formed and healthy.

X-ray photographs were taken by Dr. Thurstan Holland. In one of these, showing the left arm extended, a bridge of bone is seen stretching downwards and inwards from the neck of the radius to the upper end of the shaft of the ulna. In all probability a similar condition is present on the right side, but the position in which the arm was held while being photographed does not allow of its demonstration.

I have been unable to find any reference to a similar case. Congenital dislocations of the head of the radius have, in some few instances, been associated with bony union between the radius and ulna, but in the present case these bones appear to be normally placed.

I should like to have the views of the Society regarding the origin of this condition, and also an expression of opinion as to the most satisfactory method of treating it. Removal of the upper end of the radius, and the bridge of bone connecting it with the ulna, would appear to me to offer the best prospect of a good functional result.

(*Read April the 14th*, 1905.)

Discussion.

The Chairman (**Mr. R. C. Lucas**) asked Mr. Dun whether the muscles were normal, and whether the extension and flexion were perfect at the wrist and elbow. He thought the condition must have occurred in consequence of some nervous disturbance in early fœtal life, and that secondary ossification took place between the two bones, supination thus being rendered impossible. He hoped Mr. Dun was not of opinion that the case was beyond the reach of surgery, and that at some time he would do something to restore the use of the limb. If he was certain it was due to ossific union, it would be quite fair to cut down and remove the piece of bone which united the radius and ulna.

Dr. C. W. Chapman asked if there was any deformity in the arms in the parents. Five years ago he showed, before the Clinical Society, with X-ray photographs, a child in whom there was an absence of the ulna and deformity of both hands. He also showed the father, who had very much the same condition of affairs.

Mr. R. C. Dun (Liverpool), in reply, said there was absolute fixation; the other muscles acted normally. Flexion and extension were free at the wrist and elbow. He could not ascertain the presence of any other congenital deformity in the family.

There were two other children, who were well developed and healthy. He hoped later to do something surgically to allow of supination. The interesting question was whether it would be possible to remove the bridge of bone without excising that portion of the upper end of the radius to which it was attached. If it were merely divided, it would be very difficult to prevent reunion.

72. SOME POINTS IN THE DIAGNOSIS AND TREATMENT OF APPENDICITIS IN CHILDREN.

By H. ANGLIN WHITELOCKE, C.M.

To attempt anything like an exhaustive treatment of appendicitis in the time allotted is out of the question. I shall therefore try to direct attention to a few points which seem to affect the diagnosis and treatment of the malady as it occurs in children and young persons. To make myself clearer I have thought it wiser to read the notes of a few of my more recent cases.

In these records certain symptoms will be found to be very generally present, and it is to some of these that I now specially wish to refer. If we compare and contrast them with those usually met with in the adult, it will not be difficult to discern differences of decided clinical importance.

When called upon to attend an adult in a typical case we have usually the history of indigestion and constipation for varying periods, then of a sudden seizure of pain, at first referred to the umbilicus, with nausea and sickness. The pain is continuous, with exacerbations, the sickness not profuse, intermittent, and even subsiding later. The pain is localised in the right iliac fossa, and the bowels refuse to act. The appetite fails, the tongue becomes coated and dry, the temperature ranges from 99° to 103° F., and the pulse is accelerated. The abdomen is tense and tender to the right side of the middle line below the umbilicus. Usually these signs last for about seven days, and in twenty-one completely subside. Relapses are said to occur in 30 per cent. of cases.

The following reports are made of cases in children under 10 years :

Case 1.—R. B—, aged 5 years, was admitted into the Radcliffe Infirmary on May the 29th, 1903, with the history that he had never been ill until seven days before admission, when he was suddenly seized with symptoms of acute diarrhœa and vomiting, and complained of intense pain in the lower abdomen, with its centre of greatest intensity in the region of the urinary bladder, passing thence into the left iliac fossa. On the following day the sickness and diarrhœa continued, accompanied by tenesmus and the passing of slimy, blood-stained mucus, while the pain was transferred to the right side of the abdomen. On admission the temperature in the mouth was 102·2° F., the pulse rate 112 to the minute. A swelling could be seen and felt in the region of the appendix, which was dull on percussion.

Whilst his legs were drawn up and the belly-wall tense he complained of no pain. Two hours after admission chloroform was administered and an oblique incision made over the appendix through the skin and external oblique muscle. In separating the fibres of the internal oblique and transversalis muscles the finger slipped into a large abscess cavity. About 6 ounces of very thick fœtid pus escaped. The space had definite limitations, and firmly imbedded in its inner wall was the appendix, the tip of which, projecting freely into the cavity, was gangrenous, and contained two concretions. This was clipped off with scissors. After washing out with hot saline solution, a loose packing of bicyanide gauze was inserted and the extremities of the wound closed with sutures of silkworm gut. A strip of gauze served as the only drain. The operation was well borne, and twelve hours later, at the time of the first dressing, the temperature had fallen to normal, and never rose above 99° F. until the healing was completed. On the twenty-eighth day after the operation he was discharged well.

Case 2.—R. R—, aged 9 years, was admitted into hospital on September the 19th, 1904, having been sent in by Dr. Rice, of Steventon, Berks, who kindly supplied me with his notes:

"The symptoms in this case never pointed so definitely

to appendix trouble as they usually do. He came home from Weymouth, having been taken suddenly ill on August the 24th, with vomiting and some diarrhœa. The tenderness was never more marked over the right iliac region than other parts of the abdomen till lately. The colon was particularly tender and dilated, markedly at the flexures; there was much diarrhœa and much flatus. I imagined at one time it might be going to turn out enteric; then I leaned to colitis, the stools were so foul and slimy. The temperature was never very high, 102° F. the highest; the pulse never quick, usually 82 to the minute. There was always as much tenderness over the sigmoid as over the cæcum, and he always had pain in passing water. The temperature had been irregular for weeks and he lost flesh."

On admission he lay with his legs drawn up; his abdomen was rigid and moved badly when breathing; a distinct resistance could be made out in McBurney's point and the slightest pressure in this region caused intense suffering. The ordinary incision was made and a very foul abscess evacuated. The appendix had sloughed off and a concretion floated out with the pus. The cavity was treated as in the last case, and he made an uninterrupted recovery, leaving for home on the thirty-first day after the operation.

Case 3.—V. A—, aged 9 years, admitted into hospital December the 13th, 1904. Six days before was taken suddenly with sickness and diarrhœa, followed by acute pain all over the abdomen. The morning temperature during the illness varied from 100° to 101° F.; his bowels moved daily and flatus freely passed. He has not been sick since the outset, but has had much pain whenever he tried to pass urine and experienced difficulty in starting the act. Dr. Cruikshank, of Eynsham, under whose care he was, supplied me with the history of his having had an attack in July last of an "obscure abdominal condition, when appendicitis was suspected." His mother asserted positively that during the July attack the pain was on the left side of the belly the whole time, and that "this was what puzzled the doctor."

On admission he looked pinched and ill, tongue coated with thick white fur, breath offensive, temperature 99° F., pulse 96 to the minute, and legs drawn up. Complained that the umbilicus was the most painful spot; abdomen moved fairly in respiration, though the recti muscles were held absolutely rigid. On palpation the whole abdomen below the umbilicus was painful, the right not more than the left.

On December the 18th tongue clean, temperature normal, but severe pain at umbilicus of a dragging and cutting kind. Tenesmus constant and discharge of mucus frequent.

On the 19th laparotomy was performed across the spino-umbilical line, over McBurney's point. When the peritoneum was opened a little serum escaped but no pus. The omentum was very adherent to the small bowel. With some considerable difficulty the appendix was found passing upwards and inwards behind the lower ileum and mesentery. It was rigid, about 8 inches in length, inflamed, and firmly bound down to the front of the spine by old adhesions at the level of the umbilicus. With much difficulty and after careful separation of adhesions the tip was freed, and by degrees it became possible to clamp off the meso-appendix and separate the organ in its entire length. It was cut off close to the cæcum and invaginated into it after McBurney's plan. A small concretion was found in the bulbous extremity which doubtless was responsible for the previous attack. The usual technique was carried out, and he was dismissed five weeks after the operation.

Case 4 was that of M. H—, aged 8 years, who was admitted into hospital on March the 24th of this year and died on the same day. Two days before admission he was quite well and went to school in the morning and acted as an acolyte, as usual, at church. At noon, when at home, he was suddenly seized with pain in the lower part of the abdomen on the left of the middle line. He vomited once that evening and had diarrhœa at night.

On the 23rd pain was general over the lower abdomen, but the sickness ceased, while the diarrhœa was intensified.

No rigors. On the 24th a medical man saw him for the first time and sent him to the hospital.

On admission I saw him; pulse 145 to the minute, feeble and running; abdomen rigid, right side very tender on palpation, the muscular resistance general, the legs flexed. When asked where the pain was greatest, he put his hand to McBurney's point. There was a little dulness in the right flank. It was obvious that he had general peritonitis, so within thirty minutes I opened him in the right iliac fossa.

Some straw-coloured limpid fluid escaped which was rather offensive and came freely from the general peritoneal cavity. I did not wash out but inserted a strip of gauze and dressed the wound as gently as possible. This took place at 12.30 p.m.; at 3.30 p.m. he seemed better, but at 4.30 p.m. he suddenly became faint and vomited about two pints of blood and died almost immediately.

Post-mortem examination.—The whole abdomen was filled with a primrose-coloured, sero-purulent, foul-smelling material. The cæcum and 6 inches of the lower ileum were removed *en masse*. The appendix showed one spiral twist upon itself and for about 1 inch of its course was normal, then it became adherent to the posterior wall of the ileum, and was curled upon itself for a distance of $1\frac{1}{2}$ inches. The terminal adherent part was much inflamed and covered with purulent exudation. It had near its commencement a small perforation. On the proximal side of the perforation was a concretion.

The gastric mucous membrane was swollen, œdematous, and bloodstained, and presented small petechial sub-mucous hæmorrhages; the lungs, pleuræ, pericardium, and spleen showed petechial hæmorrhages, and the other organs cloudy swelling. This is an instance of acute fulminating or gangrenous appendicitis, with perforation and general peritonitis, the chief peculiarities being its terrible malignancy and its unusual fatal termination by hæmorrhage from the gastric mucous membrane.

It will be noticed in analysing these cases that certain unusual symptoms are noted:

(1) The presence of diarrhœa and absence of constipation in all the cases.

(2) The pain having been in each case primarily on the left side of the middle line of the abdomen, or at the umbilicus, and only becoming localised in or referred to the right side some hours after the acute onset.

(3) The vesical symptoms—nearly always pain in voiding urine, or a difficulty in initiating the act.

(4) The presence of tenesmus and the passing of blood-stained mucus in two of the cases.

In other respects the signs are similar to those pertaining in the adult.

The profession, rightly or wrongly, seems to be impressed with the belief that appendicitis is a rare and unusual disease in children. I am inclined to think that it occurs between one fourth and one third less frequently than in adults, but seems to be on the increase.

Until we realise that it is by no means a rare disease, and that we must be on the alert to recognise it in its earliest stages, I fear we shall meet with instances where the diagnosis has been missed, the treatment carried out on wrong lines, and with occasionally untoward results.

In the more severe cases it is almost impossible to ignore the possibility of acute intestinal strangulation, and where there is marked tenseness and the passage of mucus intussusception must be differentiated. I have known the pain in the bladder lead to the diagnosis of vesical calculus and the organ sounded for stone.

Perhaps the most general interpretation of the symptoms is simple intestinal colic, a symptom so common in growing children that it is not altogether surprising that strong aperients are so frequently exhibited, to the marked detriment of the later treatment. Mistakes will be less likely to be made if proper attention is given to a more thorough interpretation of the signs and symptoms.

Why we should almost invariably find constipation in the adult, while it is very often absent or gives place to the opposite condition in the child or young person, I cannot explain. Once upon a time I used to regard constipation as

a conservative process, a provision of Nature for securing rest for the inflamed and irritated intestines, yet in children it does not seem to serve this purpose. And observing this fact, may it not in a measure explain the opposite views held by the schools as to the treatment of inflammatory affections of the intestines? While most persons cease to use purgatives and lock up the bowels, others urge the immoderate and continued use of purgation. Be the cause what it may, the fact remains that diarrhœa is very prevalent in children with appendicitis.

Then as to the pain being referred to the *left* side early in the attack. Is this an instance of purely so-called referred pain, with only a remote and indirect anatomical relationship, or is it due rather to the direct anatomical situation of the particular appendix at fault?

For while we know, on the one hand, from the researches of Head,* that referred pains have in most cases a direct relationship to visceral disease, so, on the other hand, we know that the appendix may be anatomically situated in many and various positions. According to Testut it may be found:

(1) Passing upwards and inwards behind the ileum.

(2) Over the brim of the pelvis and external iliac vessels.

(3) Downwards towards Poupart's ligament.

(4) Upwards and along the outer margin of the cæcum and ascending colon.

(5) Directly up, behind, and concealed by the cæcum.

Head records the case of a boy, aged 16 years, whose right undescended testis was injured, and who suffered pain in the tenth dorsal segment, more on the left side than on the right.

Some such interpretation as Head's may be suspected, yet I lean towards the latter view as the more likely to be helpful. The pain at the umbilicus in Case 3 was doubtless due to the adhesions of that abnormally long appendix to the lumbar spine; and in two cases of female children, aged 11 years and 12 years, with whom I had to deal, and in whom the pain was referred chiefly to the left side and to the bladder, the abscesses were in Douglas's

* Head, "Referred Pain," 'Brain,' vol. xvi, 1893.

pouch and the appendix adherent in the pelvis. In cases of abscess it is often very difficult when operating to locate the situation of the organ, nor do I think the effort an advisable one to make, and for obvious reasons.

The vesical pain or inconvenience is sometimes due to a distended condition of the viscus, for it is often materially relieved by catheterisation, whilst in most cases it is probably due to the direct extension of the inflammatory process to the false ligaments of the bladder. In attempting to make a diagnosis the various conditions above mentioned must be taken into consideration, and it is a wise plan always in children to make a gentle bimanual examination, under an anæsthetic if necessary, with one finger in the lower bowel. When the true pelvis is involved, and in a certain number of cases even where the appendix is in its most usual situation, the swelling may be felt with comparative ease by this manœuvre.

As to treatment, I will refer merely to those cases requiring surgical measures, such as the gangrenous, purulent, relapsing (either from the presence of concretions, adhesions, stricture, or partial obliteration), and the rarely found foreign bodies.

The milder or catarrhal forms rarely fall to the surgeon and are best left to the care of the physician. Purgatives should never, in my opinion, be used; enemata are always safer. Opium should seldom be given; it masks the symptoms and increases the general distension.

As to operation. In the serious cases, as the gangrenous or fulminating, the sooner laparotomy is undertaken the better. Where there is suppuration I freely open, wash or wipe out the cavity with gentleness and care, removing concretions and sloughs, but do not specially seek to find the appendix. I always use a strip of gauze as a drain. It is quite efficacious, and stimulates the sealing-off tendency of the peritoneum. A glass or rubber tube is prone to perforate the already inflamed and rigid gut and give rise to a fæcal fistula after a dressing is applied; for the pathological principle that constant pressure causes atrophy is applicable here as elsewhere.

Children do not bear laparotomy as well as adults, and the operation should be performed quickly.

During the years 1901 to 1905 inclusive I have operated on 23 cases of children under 12 years, 6 females and 17 males, with a mortality of two (both fulminating cases, one of which is recorded as Case 4).

In 6 cases the appendix was wholly or partially gangrenous; in 13 there was an abscess; in 3 a relapsing condition, the appendix in each case containing a concretion; 1 partially obliterated, without a solid concretion but with fluid contents.

Two children, one in hospital and the other in private practice, were under 4 years; both of these had abscesses, were operated upon, and ultimately did well.

(*Read April the 14th,* 1905.)

Discussion.

Dr. Percy Lewis (Folkestone) said an important point was whether such cases should be washed out at the operation. He thought the author had not sufficiently separated those cases. Where there were adhesions and a limited abscess the washing out should be done gently; but in the fulminating form, where there was pus spreading in all directions, one could not do better than wash out most thoroughly. Since he had done that he had obtained very much better results than when he simply put in a tube. Last summer he had a girl, aged 14 years, with fulminating appendicitis, and within twenty-four hours of the onset the abdomen was opened and afterwards washed out, every part of the abdomen, including round the liver, and an opening was also made in the middle line and a tube was put in Douglas's pouch. She got quite well, and was out driving in three weeks' time. He thought she would have died from the amount of poison in her if she had not been thoroughly washed out. He protested against the dictum that opium should never be used in such cases; it would be very hard on the patients, and he thought anyone who had appendicitis would want opium. It was stated it would mask the diagnosis; if so, the diagnosis must be improved. In the old days, when the condition was spoken of as peritonitis, a large number of cases were cured by opium and blue pill; and no doubt it was a very excellent treatment, for the opium put the inflamed parts at rest, and he thought there could be no physiological objection to it.

Mr. Lockhart Mummery said Mr. Whitelocke had brought out one point not generally mentioned, namely, that in children

the tendency was towards the fulminating variety rather than the catarrhal. The majority of cases in children in which there was gangrene were those of acute abscess, and that showed that one was not justified in waiting to see whether the attack would pass off in children with the idea of operating in the quiescent stage. Directly appendicitis was diagnosed in a child under 12 years of age the abdomen should be opened. He knew of two recent cases, not his own, in which operation was deferred until it was too late. His belief was that it was the best practice to wash out the abdomen thoroughly, especially where there was general peritonitis. Another sound method of dealing with general peritonitis was to put two large tubes into the pelvis, one on each side, through small incisions, and irrigate continuously through those tubes or every half-hour with saline solution, and nurse the patient in a semi-sitting posture. That constituted a very effectual means of draining those cases. The results from that method seemed to be better than by any other, and the necessary operation was extremely small.

Mr. R. C. Dun (Liverpool) said his experience bore out Mr. Whitelocke's as to the frequency of diarrhœa and bladder irritation in appendicitis in children compared with adults. In several of his cases there was complete retention of urine. He thought it doubtful whether there was a tendency for children to have a more serious type of appendicitis than adults; the idea was probably due to the likelihood of appendicitis occurring in children not being sufficiently before the profession, with the result that the slight attacks were missed. Practically in all his cases of abscess or perforation careful inquiry had elicited evidence pointing to previous attacks. Frequent attacks of colic in children might be referable to the appendix. He would ask Mr. Whitelocke his experience as to the value of the blood-count in the diagnosis of pus-formation in children. He had found in all cases in which there were more than 15,000 leucocytes to the cubic millimetre that pus was present. He agreed that the removal of an appendix which was firmly adherent to the wall of an abscess was associated with great risk, but in two cases in which he was compelled to leave the appendix symptoms of appendicitis had recurred after the abscess cavity had healed up. In both of them a second operation had to be performed for the removal of the appendix. That, however, was not associated with so much risk as attempting to remove a very adherent appendix when treating the abscess. The prognosis was extremely grave in gangrenous appendicitis associated with general peritonitis. The only two cases which he had met with proved rapidly fatal. He thought washing out the abdominal cavity with saline solution was the best form of treatment. The plan adopted was to put a tube in the appendix incision, another through an opening in the middle line, going into Douglas's pouch, and two tubes through the flanks, irrigating through all four tubes.

Dr. C. W. Chapman said he thought it would be found that the children who suffered from colic were in the habit of bolting their food. He heard of a large institution in which the children were allowed only half an hour in which to wash their hands, dress and have dinner, and get back to work. He wrote to the doctor about it, and suggested that they should have plenty of time for their meals in order to masticate their food thoroughly.

The Chairman (**Mr. R. C. Lucas**) said the subject was sufficiently large to occupy a whole evening, so he would not attempt to cover it, but would offer one or two remarks. No doubt colic in children was often due to indigestion, as Dr. Chapman mentioned, but when that had been treated for some long time without the pain ceasing, although the pain was chiefly in the central line, it was important that a surgeon should carefully examine the region of the appendix. A case which had been under treatment for many months, and in which the diet had been most carefully regulated, was sent to him a few months ago. He could feel the appendix rolling under his hand, and though there was no very great tenderness, he recommended that it should be cut down upon. He did so, and some serum escaped from the peritoneal cavity. He then found an appendix six inches in length, with only one adhesion, at its end, and that had sufficiently accounted for all the symptoms. The appendix was removed, and the case thereby cured. He thought there was an explanation for the bladder irritation being more common in children than in adults. The bladder was much more an abdominal organ in a child, and therefore inflammation occurring in the lower part was more likely to affect the bladder in a child than in an adult. He would like to hear why, in the last case narrated by Mr. Whitelocke, he did not remove the appendix. He (Mr. Lucas) thought it unwise to perform an incomplete operation unless the patient was actually *in extremis*. He agreed that the washing out of those cases was an advantage. His experience was that if the patient was very low the best thing was to fill the abdomen with saline fluid. So far from it being a depressant, the saline fluid raised the pulse at once, especially in general peritonitis.

Mr. R. H. Anglin Whitelocke (Oxford), in reply, thanked those who had criticised his paper. In reply to the Chairman (Mr. R. Clement Lucas), he did not remove the appendix in his last case because the omentum was matted to the small gut, the coils were matted together, and the patient was *in extremis*, the pulse being almost imperceptible. He did not die from shock apparently, but from the hæmorrhage from the stomach. He had heard of this symptom in only one similar case—one of acute septic peritonitis with hæmorrhages under all the mucous membranes of the body. He agreed that washing out of the abdomen should be carried out in adults, but he had seen in the hands of others cases which he felt sure were lost through too much

washing out. It was true the pulse improved as soon as the warm fluid entered the abdomen, but it failed again, often suddenly, in a few minutes' time. He had made up his mind not to wash out the abdomen in children at the time of operation, as a rule, though it might be done subsequently. He practised the new method of draining in adults. It must be remembered that time was the important element in the case of children, and they would not stand much pulling about, whether for appendicitis, strangulated hernia, or any other acute condition. He had seen a child die as soon as the coils of intestine were pulled down and exposed. The use or abuse of opium would always be a prolific subject for debate, but the more he saw of abdominal surgery the more he was convinced that opium was deleterious. He had discussed the matter with Mr. Mayo Robson, who said he had abandoned the use of opium; where anything of the kind was necessary he resorted to aspirin or something else. The condition of the pulse was a very great indication as to whether one had to deal with an acute gangrenous condition. It was shabby and rapid, and when it began to fail laparotomy, if only for purposes of exploration, should be performed. He agreed with Mr. Dun's explanation of the greater frequency with which fulminating cases of appendicitis were seen in children: the fact was the surgeon did not see the milder cases; they were treated by the physicians, and it was only when untoward symptoms occurred that the surgeon was called in. He generally had a blood-count done in adults, but not in children. In relapsing forms in adults the blood-count gave an excellent idea as to suppuration. He would see that his house-surgeon did the count in all doubtful cases in future.

73. CASE OF EXOPHTHALMIC GOITRE IN A CHILD AGED 3½ YEARS.

By CHARLES BATCHELOR.

The patient was the only child of healthy parents, in a good position of life. There was no family history of insanity or neuroses. She was perfectly healthy until she had whooping-cough in the early months of 1901.

In June of that year, at the age of 3 years and 1 month, it was noticed that her eyes were prominent, and she had attacks of passion, which had never been pronounced before.

On her return from the seaside in September, I found that she had very definite exophthalmos, with decided enlargement of the thyroid, the right lobe being larger than the left. There were no tremors, but the heart's action was rapid, and the attacks of passion, with loud screaming, were much more frequent.

In March she had an attack of influenza and pneumonia, and was in a most critical condition for several days.

During the summer the exophthalmos became even more pronounced. The child was generally breathless on exertion, and the heart's action always rapid (120–140 beats per minute), with marked pulsation of the carotids, but there were no murmurs, and never albuminuria. The prominence of the eyes varied slightly from time to time, but the eyelids always closed during sleep. Von Graefe's sign was present. I was never able to discover any tremors.

In 1904 she had two or three attacks of sickness, which produced great prostration, and on September the 24th she was attacked with diarrhœa, followed by uncontrollable vomiting, and she died from exhaustion on September the 26th at the age of 6 years and 4 months.

(*Read April the* 14*th*, 1905.)

Discussion.

The **Chairman** (**Mr. R. C. Lucas**) said it was a very remarkable case, and it was very rare for a case of exophthalmic goitre to occur at so early an age. The whooping-cough seemed to have occurred immediately before the disease commenced. He did not know whether any connection could be suggested between the two. There was a gradually increasing exophthalmos, as shown by the photographs.

Mr. Sydney Stephenson remarked that to say exophthalmic goitre was very uncommon in children was to utter a truism. At the same time, Mr. Batchelor had established a record even for that Society. Members would remember that three cases had been shown of exophthalmic goitre, two, curiously enough, in children of 12 years of age; two of them were girls and one boy. But the Society's records hitherto had contained no instance of Graves' disease in a younger child. That seemed to be one more reason why the profession should extend a large recognition to the Society, that it should preserve and foster the publication of

such interesting cases which might otherwise be lost to medical science.

Dr. G. A. Sutherland said the record of the case formed a valuable addition to the 'Reports' of the Society. He considered that it was the earliest case of the kind on record. He was much struck by the rapid course of the illness, which had a duration of only three years. That illustrated a not uncommon experience, that some diseases which were more or less chronic in adults tended to run a very rapid course when they developed in early life. He understood there was an absence in this case of acute mental symptoms, which in this disease were usually present in fatal cases. Then there was the feature of its termination with acute abdominal symptoms. In exophthalmic goitre it was common to have those abdominal attacks with profuse diarrhœa, which resisted treatment. Very few people had an opportunity of seeing a fatal case of exophthalmic goitre, because it was an affection which persisted for years, and if death followed it was usually from some intercurrent disease.

Mr. George Pernet asked whether there had been any skin changes in the case.

Dr. Thomas W. Parkinson asked whether there was any history of myxœdema.

Dr. W. Ewart asked whether there was any tremor in the case, and whether it might be concluded that the patient did not display any remarkable amount of muscular weakness.

Mr. Charles Batchelor (Staines), in reply, said such a case was so rare that he felt it his duty to present the record of it to the Society. There was no history of myxœdema in the family. There were no skin changes, such as pigmentation or a rash. Neither were there tremors or muscular weakness. The last photograph was taken ten days before death.

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74. VERRUCOSE ENDOCARDITIS OF THE AORTIC VALVES.

By EDMUND CAUTLEY, M.D.

Infective endocarditis is by no means common in young children, and it is still more rare to find the peculiar chronic verrucose condition which was present in this instance. The case was that of a boy, aged 8 years, who was admitted into the Belgrave Hospital for Children for supposed meningitis on March the 6th, and died on March

the 17th, 1905. The family history contained no facts of importance, and his past history was excellent, except for the suggestive point that he had been in St. Thomas' Hospital some years previous for "weak heart."

Three weeks before admission he received a blow on the right eye through a fall on a bench at school. He was taken home and put to bed for a few days, then he was allowed up, but during the last ten days he had been again confined to his bed. The symptoms, since the injury, consisted of constant vomiting, anorexia, constipation, variable headache in the right frontal region, vague pains in the chest and abdomen, and fever. Two days ago the temperature reached 103·6° F. There was no delirium. On admission he was conscious, drowsy, and rather irritable. There was a little duskiness of the nose and lips, but no clubbing of the extremities. The apex of the heart was in the fifth space, nipple line, the impulse rather exaggerated, and the dulness a little increased. A loud systolic murmur was heard all over, loudest at the base, with its point of maximum intensity in the second left interspace near the sternum. It was conducted into the neck on both sides, into the axilla, and heard in both interscapular regions. The pulse was of fair volume, about 100 per minute. A blood-count yielded 5,800,000 red cells and 8500 white cells. There was no abdominal distension. The liver and spleen were palpable and the splenic dulness enlarged.

March the 7th.—Good night, still irritable, more drowsy. Occasional headache. No delirium. Anorexia, no vomiting, normal stools. The knee-jerks were absent and Babinski's reflex was present.

March the 10th.—The temperature has ranged between 100·6° F. and 105·8° F., being very irregular in character. Pulse 120, rather small. Has vomited twice in the last day.

March the 14th.—The temperature maintains its irregularity and has been up to 104·6° F. The murmur is now definitely loudest in the aortic region. The spleen is larger. A blood-count yielded 12,000 white cells.

March the 17th.—For two days there has been frequent

vomiting. Last night patient became very excitable, but this morning he collapsed, turning a very bad colour. Two hours later he became unconscious, and he died in a state of coma in another two hours.

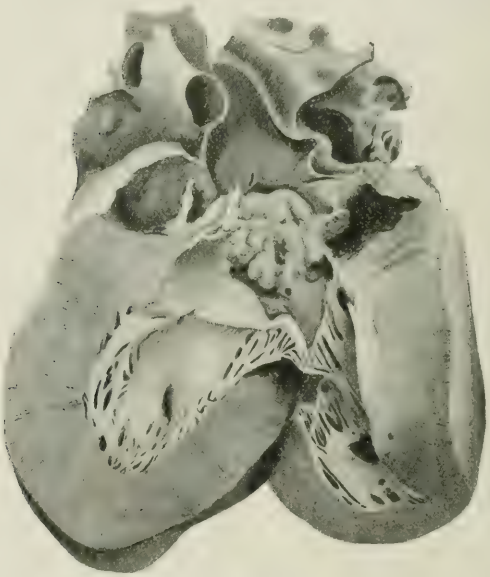
The diagnosis of this case was peculiarly difficult on account of the very definite relationship of the onset of the illness to injury. It was, however, perfectly clear that there was more than a meningitis, even if there was any meningeal affection at all. There were no signs sufficiently definite to suggest typhoid fever. The presence of a basal cardiac murmur might have been only a coincidence, but in view of the character of the fever, the enlargement of the spleen, and the absence of marked meningeal symptoms I regarded the case as one of infective endocarditis, secondary to a valvular lesion which was probably congenital. The distribution of the murmur at first suggested a pulmonary stenosis, but there were none of the physical signs usually present in this affection. Later, the distribution of the murmur and the evidence of hypertrophy of the left ventricle indicated an aortic affection, a comparatively rare form of valvular disease in children as a single lesion.

On post-mortem examination an excess of fluid was found in the cerebro-spinal canal. In the pia-arachnoid, scattered over the cortex and base of the brain, were a few small isolated plaques of thin purulent lymph. The pericardium was distended with recent effusion of serum and blood. The heart was larger than normal, the enlargement being due to hypertrophy of the left ventricle, and moderate dilatation of the left auricle. In the base of the wall of the left ventricle there was a recent infarct, the size of a small walnut, containing very soft blood-clot, commencing to break down. The aortic valves were converted into irregular warty masses, showing no trace of the original valve structure, the warty structure being very firm in texture. Above the valves the aorta was considerably dilated, and contained a large, flat, circular patch, about the size of a shilling, of inflammatory tissue, and looking like a large sessile wart. The hæmorrhage

into the pericardium had taken place through the thinned wall of the aorta. The spleen was enlarged, and contained a recent infarct. All the other organs were normal.

It is doubtful what was the cause of the primary affection of the aortic valves. From the fact that only these valves were affected, and that there was neither a family nor a past history of rheumatic affections, and that there

Fig. 13.



was a past history of "arch heart," I am inclined to the view that the mischief was congenital in origin. That congenital aortic stenosis can occur is recognised. Two such cases have come under my notice, one of which I showed at a meeting of the Medical Society of London in 1899 and the other before this Society ('Reports,' vol. i). Fisher also showed a specimen of the lesion before this Society ('Reports,' vol. ii, p. 16). Friedmann reported a case in which the murmur was heard at the age of 4 months.

Infective endocarditis of the aortic valves is naturally

most uncommon; for infective endocarditis is uncommon, and that the aortic valves alone should be affected is still more rare. Usually when it occurs it is a secondary affection of the mitral valves. In one case under my care the child had pulmonary stenosis and regurgitation, with secondary infection. Her age was 15 years.

(*Read April the 14th,* 1905.)

Discussion.

Dr. C. W. Chapman asked whether it was possible that the injury mentioned had something to do with it. He had a case recently in the person of a porter on the L. & N. W. Ry. who had an injury to his shoulder resulting in suppurative mischief. Ever since the accident he had had difficulty in breathing, and he eventually died of septic endocarditis.

Dr. W. Ewart, in view of the use of the term "verrucose," asked whether there was any loose fibrin or pedunculated vegetation. The symptoms were those of infective malignant endocarditis; and yet it was remarkable that there was not that usual accompaniment of it, the loose fibrin, bearing micro-organisms in quantity. That made the specimen a most interesting one. The hardness of the knobby formation justified the use of the term "verrucose," but the size of the masses was a little against it. The usual form of verrucose valvular endocarditis was that in which the deposits were very firm, sessile, and smooth, not roughened or ulcerated, but as a rule small. There was the same hardness, and apparently the same absence of ulceration or pedunculation; but the "warty" outgrowth was of considerable size. Had a microscopical examination been made?

Mr. William H. Vickery (Newcastle) asked whether it was possible that the original lesion of the valve was due to congenital syphilis.

Dr. Edmund Cautley, in reply, said it was possible that injury might have had something to do with causing the secondary infection, but it was difficult to assign a source of infection in most of those cases. He agreed with Dr. Ewart's remark about the nomenclature. Verrucose struck him as being the best name, because of the hard, somewhat warty condition of the valves and the structure in the aorta like that of a large sessile wart. The alteration of the valves was very peculiar. The growth was as hard at the time the heart was opened as it was after the specimen had been kept in formalin. He could not detect loose fibrin nor the source of the embolisms which had been carried into the heart-muscle and into the spleen. There was sufficient evidence to enable one to speak of it as infective endocarditis

because the aorta was softened sufficiently to allow a leakage of blood into the pericardium, and the infarct in the wall of the heart was breaking down. It was a mild form of infective endocarditis grafted on to a warty condition of valves, which had been in existence for some time previously and could not have been produced in the five weeks which elapsed after the injury. There was no evidence in the patient of syphilis and there was no history of it in the family. No microscopic section had been made.

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75. A CASE OF CONGENITAL STRICTURE OF THE ŒSOPHAGUS.

By R. C. DUN, C.M.

In October, 1903, a boy, aged 18 months, was seen in the Out-Patient Department of the Liverpool Infirmary for Children, and the following history ascertained:

Three weeks before he was taken to hospital he suddenly and without apparent cause began to vomit everything, both solid and liquid. The child had been under medical treatment since the vomiting set in, but various changes of diet and drugs had not led to any improvement. No attempt had been made to pass œsophageal bougies. Rapid wasting had occurred since the onset of the vomiting. The previous health had been good. The mother had no suspicion of a foreign body having been swallowed. There was no evidence of a tubercular or syphilitic history, and nine other children were healthy and well formed.

The child was pale and thin. He weighed 18 lbs. 4 oz. The heart, lungs, and spine were normal. No signs of tubercle or syphilis were present, and no obvious congenital deformity was noted. The boy was ravenously hungry; he swallowed milk greedily in small sips and retained it, but would not attempt a long drink. Bread and butter was returned, with slight retching and alkaline in reaction, a minute or two after several mouthfuls had been swallowed. No swelling was noted about the neck.

Various sizes of olivary œsophageal bougies were passed;

with all obstruction was met with 7½ inches from the teeth. No foreign body was felt, and examination with the fluorescent screen was negative.

A provisional diagnosis of congenital stricture of the lower third of the œsophagus was made.

Owing to the wards being overcrowded at the time, and as the child was apparently able to take enough milk for his requirements, it was not until two days later that he was admitted into hospital. The mother then reported that the boy had taken his milk well since his visit to the Out-Patient Department, that none of it had been returned, and that he had eaten and retained "a gingerbread" before leaving home the same morning.

On admission, October the 17th, 1903, any solid food given was returned as before described. Milk swallowed in small quantities, one to two teaspoonfuls at a time, was retained, but larger drinks regurgitated from half to two minutes after being swallowed. On the third day in hospital the child was able to retain milk pudding, given slowly and in small quantities, but after twenty-four hours this improvement ceased, and the pudding began to be returned almost immediately after being swallowed. Ten days later, on a diet of milk alone, the weight remained stationary at 18 lbs. 4 oz. Under chloroform, bougies of various materials and sizes were passed. All the more rigid instruments were arrested at a point 7½ inches from the teeth: some of the finer and more flexible appeared to curl up when the obstruction was reached, but none could be felt to engage in the stricture. The stomach was not entered.

On November the 4th, five days after the passing of bougies, milk began to be returned after about half an ounce had been swallowed, and the child was losing weight. Nutrient enemata every six hours, with a daily washing out of the bowel, were ordered. The third enema, given on the evening of November the 25th, was returned, and was followed by the development of an intussusception which could be felt through the abdominal wall and on rectal

examination. The intussusception was promptly reduced by Dr. M. White, then senior house-surgeon, by means of an olive-oil injection. Later in the evening I saw the case. The œsophageal obstruction appeared to be complete, and it was obviously impossible to attempt the continuance of rectal feeding. I therefore opened the abdomen, made an examination to assure myself that the intussusception was completely reduced, and performed a gastrostomy after Frank's method. The child was fed on the operating table and stood the operation well.

Six days later (November the 11th) there was a gain in weight of 2 oz., the feeding being entirely through the gastrostomy opening. The stomach capacity was small, two ounces constituting an adequate feed; with this amount no regurgitation through the fistula occurred.

The œsophageal obstruction varied in degree. The child was frequently tried with small quantities of milk by the mouth; sometimes a little would be retained, but as a rule when about half an ounce had been swallowed it was returned. The saliva usually found its way into the stomach, but from time to time several ounces of mucus and saliva, always alkaline in reaction, would be brought up. This would occur for a day or two, and then not be noted again for several weeks.

On November the 19th part of a gastrostomy feed which had just been given was returned through the fistula, about 3 ounces of mucus and saliva were vomited and another intussusception developed. This was again immediately reduced by fluid injection.

For another month the child lived on his gastrostomy feeds, at rare intervals retaining a few tablespoonfuls of milk taken by the mouth and occasionally vomiting mucus and saliva. On two occasions bougies were passed, under chloroform, but without reaching the stomach.

December the 14th.—The weight had fallen to 17 lbs., a loss of 1 lb. 4 oz. since admission into hospital. I decided to perform a gastrotomy and attempt to reach, and if possible pass instruments through, the stricture from below.

It seemed probable that the obstruction would be more accessible by this route than from a cervical œsophagotomy.

The stomach was opened to the left of the gastrostomy fistula and without interfering with it, the finger was introduced, and the cardiac orifice gradually dilated. The obstruction could not be reached by the finger, but a fine bougie passed along its side was arrested half an inch beyond the tip of the finger. Attempts to pass fine curved forceps, bougies, catheters, etc., through the stricture from below all failed, and the opening in the stomach had to be stitched up without any advantage having been gained. The child was none the worse for the operation; in fact, a fortnight later (December the 28th) he had gained 8 oz. in weight.

Early in January, 1904, the boy developed bronchitis, and subsequently broncho-pneumonia, and the general condition became very grave. Nutrient enemata were again tried, and were retained. The weight fell to 14 lbs. 12 oz. on February the 18th.

From this date slow improvement was noted. On April the 9th the child swallowed and retained 10 teaspoonfuls of milk, on the 10th half an ounce, on the 11th three ounces. Gradually increasing quantities given by the mouth were now retained until by April the 19th three ounces of milk thickened with farinaceous food were being given four-hourly. The gastrostomy feeds and enemata were slowly diminished in number and quantity and were entirely given up on May the 24th. An attempt was made to pass bougies through the stricture on May the 10th, but without success. On May the 30th, 1904, the child was sent to the convalescent home, being fed by the mouth only and with a weight of 17 lbs. 8 oz.

Ten days later (June the 10th, 1904) the child was sent back from the convalescent home on account of his having returned several feeds, but under observation in hospital up till June the 21st he took fluids well by the mouth. Then tightening of the stricture appeared to occur, and gastrostomy feeds every six hours had to be given to supply sufficient nourishment. Five weeks later (July the 27th) all

mouth feeds were returned and direct stomach and rectal feeding had to be resorted to. In spite of numerous alterations in the nature of the food thus supplied, a steady diminution in weight took place, and it had fallen to 14 lbs. 9 oz. on October the 18th, when the child developed measles and was sent to the workhouse infirmary.

For about six weeks the boy was in that institution, and was then sent home. I did not see him again for a period of over five months (April, 1905), when he was again taken to the Out-Patient Department of the Children's Infirmary. While at home nothing given by the mouth had been retained. The child was extremely emaciated; he weighed 12 lbs. $10\frac{3}{4}$ oz., and was being fed entirely through the gastrostomy opening. For about a month there had been some leakage of gastric juice, giving rise to eczema; but before that the valve-like nature of the opening had effectually prevented any escape of stomach secretion.

Three weeks later, *i. e.* fifteen months after I first saw the child, I heard that he was dead, and obtained permission for a post-mortem examination.

The œsophagus, trachea, thoracic viscera, diaphragm, liver, and stomach were removed together. There was no evidence of mediastinal inflammation. The œsophagus was easily separated from the trachea, left bronchus, and aorta. The specimen shows a marked diminution in the size of the œsophagus at a point one inch above the diaphragm. The constriction is short and sudden. The œsophagus above it is larger and thicker walled than it is below, but no pouching of the tube above the stricture is present. The œsophagus has been opened along its posterior aspect, leaving the stricture intact. The lumen of the constricted portion is very narrow; it just allows the passage of a small probe. The internal longitudinal measurement of the stricture is a quarter of an inch. No other abnormality of the alimentary canal was present.

This case of congenital stricture of the œsophagus*

* *Vide* Editorial, "Congenital Stricture or Occlusion of the Œsophagus," 'British Journal of Children's Diseases,' August, 1904, p. 364.

closely resembles two others recently reported—one which was communicated by Dr. Bertram Rogers to this Society at the meeting held at Bristol in June, 1904, and published in the 'British Journal of Children's Diseases' in September, 1904; the other published in the 'Lancet,' January the 7th, 1905, by Dr. Whipham and Mr. Fagge.

In all three cases the position of the stricture has been the same, viz. in the lower third of the œsophagus between the cardiac orifice and the point of crossing of the left bronchus. In none of these cases have the symptoms appeared before the twelfth month.

In the present case the stenosis would seem to have developed more rapidly than in those previously reported, as three weeks after the onset of symptoms no instrument could be passed into the stomach, and a fortnight later complete obstruction to fluids occurred. The varying degree of the obstruction is also a notable feature.

Regarding the treatment of impermeable congenital stricture of the œsophagus, it is evident that life may be prolonged—if, indeed, that be desirable—by means of a gastrostomy, and that this is not necessarily followed by a leakage of gastric juice and skin-irritation. During the course of this case I considered the advisability of attempting to reach the lower part of the œsophagus through the mediastinum, but the general condition of the child never warranted such an operation. From an examination of the specimen obtained post mortem it does not appear likely that dilatation of the stricture, or a plastic operation for its relief, would have been possible by such a procedure.

No adequate explanation is as yet forthcoming to explain the formation of congenital stricture of the lower portion of the œsophagus. (*Exhibited April the 14th*, 1905.)

Discussion.

The Chairman (**Mr. R. C. Lucas**) regretted that the Society had not a museum into which such interesting specimens could be placed. The Society was looking forward to having a place

of its own, with library and museum, and when that time came the museum would be an interesting feature.

Dr. T. R. Whipham remarked that the interesting case just brought forward was a valuable addition to the list of cases of this rare abnormality. The case reported in the 'Lancet' was under his (Dr. Whipham's) care about a year ago, and having searched the literature at that time, he found only six previous cases which were at all analogous. These cases seemed to be comparable in some ways to those of hypertrophic stenosis of the pylorus. The latter condition was until recently more or less unrecognised, but of late years numerous cases had been diagnosed and operated upon, with a certain amount of success. He believed that in future more cases of congenital stricture of the œsophagus would be brought before the profession. The interest of these cases lay first in their origin, and secondly in the method of treatment. Accepting the present views concerning the development of the upper part of the alimentary canal, he could not explain their origin. The strictures higher up might be due to excessive narrowing of the fore-gut where it contracts to form the upper part of the œsophagus, or to a failure in the process of channelisation of the column of hypoblastic cells from which the cervical portion of the œsophagus is primitively derived, but whether that was applicable to strictures lower down in the œsophagus it was difficult to say. No treatment hitherto had proved of any avail. Dilatation was first tried in his case so as to avoid, if possible, subjecting the patient to the effects of gastrostomy throughout life. Catheters of different sizes were made with a central terminal opening through which the bulbous end of a wire stylet projected, the wire being long enough to come well beyond the mouth when the catheter was passed into the stomach. A small-sized catheter was first passed through the stricture and then withdrawn, leaving the stylet *in situ*. Over this larger catheters were successively introduced. The stricture was dilated up to a No. 11 catheter, but during the procedure the œsophagus was, unfortunately, perforated, with the inevitable result that death occurred. Whether the results of dilatation were likely to be permanent he left it for the surgeons to decide.

Dr. Edmund Cautley thought there was scarcely justification for comparing the condition with hypertrophic stenosis of the pylorus. There did not seem to him to be any hypertrophy in the present case, but merely a simple stricture of the lumen of the œsophagus. Was it certain that a case of that nature was congenital? Was it not possible that such a stricture might result from slight local injury?

Mr. R. C. Dun (Liverpool), in reply, thought dilatation was the best treatment if bougies could be got through the stricture. But in the present case he had failed after many attempts to

treat the stricture from the mouth; therefore he was forced to either let the child die from inanition or perform a gastrostomy. He supposed such a condition might arise from some slight form of injury or irritation frequently repeated. It seemed strange that if these strictures low down in the œsophagus had a congenital origin the onset of symptoms should be so long delayed. In no case had signs of obstruction occurred before the twelfth month. He did not think anything was definitely known as to how the condition arose. The constant position of the stricture in the few reported cases favoured the theory of congenital origin.

76. SILVER DEPOSIT IN CORNEA.

By SYDNEY STEPHENSON, C.M.

Nora R—, aged 4 years. First attendance North-Eastern Hospital for Children, May the 5th, 1904.

Child brought to hospital with considerable photophobia of left eye; found to be due (after ethyl chloride had been given) to a fascicular keratitis involving the upper outer quadrant of the cornea. The clinical picture was the usual one, viz., a small, greyish, raised crescent lying in the substance of the cornea, with a leash of red vessels running to it from the nearest point of the limbus. No eczema and no phthisis said to be present on either side of the family. The child was thin and white.

The condition showed no signs of improvement under the common remedies, as yellow ointment, with and without atropine, atropine drops, and syrup of the phosphate of iron. Accordingly, on November the 3rd, 1904, under ethyl chloride, the ulcer was touched with solid silver nitrate. An ointment of argyrol (gr. v), atropine (gr. j), and vaseline (ʒij) was prescribed for home use three times a day. On November the 17th the condition of the eye was as follows: No redness. A dense greyish-white deposit, probably of silver chloride or albuminate, is present in the site cauterised a fortnight ago. The eye remained

free from irritation, and the last note on the condition, made December the 1st, 1904, was as under—"Left eye: Lying in the upper, outer quadrant of the cornea is a patch, 5 mm. by 3 mm., of roughly quadrangular shape. It is very slightly raised, and surrounded by a narrow zone of opacity. No redness of the eye.

(*Exhibited May the 5th,* 1905.)

Discussion.

Mr. Ernest Clarke thought the author was unfair to himself in stating that the result obtained was due to his own fault, because for many years the use of solid nitrate of silver had been one of the recognised methods of treatment in such cases. The case was interesting as showing what might happen, and because it warned practitioners against the use of solid nitrate of silver.

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77. CASES OF COMBINED AORTIC AND MITRAL DISEASE.

By F. J. POYNTON, M.D.

Case 1.—E. C—, male, aged 13 years. Four attacks of rheumatic fever. Each attack began with arthritis and sweating.

Under observation first in July, 1901. At that date there was definite aortic and mitral disease, and he passed through his third attack of rheumatic fever. Through the latter half of 1901, during 1902, 1903, and 1904, was under observation and escaped any further attack until October, 1904; since then another attack, making the fourth.

This case illustrates the combined lesion and the pallor, and also the gradual shifting of the aortic murmur from the left toward the right of the sternum. At 8 years of age the diastolic murmur was audible almost entirely to the left of the sternum, now it is plainly audible down the right margin of the sternum. The case also illustrates that

the great danger in this condition is another attack of rheumatism, and not a mechanical failure of compensation. Even at his best the boy has been too nervous to attend school.

Case 2.—S. F—, aged 7 years. The parents are foreign and the history of the illness imperfect. Apparently the child has suffered from chorea off-and-on for two years, and there is a vague history of heart-disease of some duration. First under observation in October, 1904.

Then there was great cardiac dilatation, a well-marked aortic diastolic murmur, audible loudest in the second left intercostal space, and well-marked mitral disease. The child improved greatly under treatment, and gradually the aortic murmur disappeared, and in January, 1905, could not be detected. There is now a well-marked double mitral lesion, but although the aortic second sound is faint, no definite aortic murmur can be heard, unless perhaps in the third left intercostal space.

This case illustrates the gradual disappearance of an aortic murmur.

Case 3.—J. N—, aged 11 years. Three years ago chorea. August, 1904—Sore throat, followed by rheumatic fever and heart-disease. Under observation since February, 1905, then active carditis and nodules. The double lesion is exceedingly well marked, and the heart quite remarkable for its enormous size. Special features: (1) the remarkably good colour of the face; (2) the extensive valvular lesions; (3) the enormous heart.

The nodules have disappeared. The pericardium is probably adherent.

Case 4.—W. P—, aged 7 years. Good health until November, 1904, then a very severe attack of rheumatic fever, with vomiting and abdominal pains, pericarditis, endocarditis, and chorea. Slow recovery and definite evidence of aortic and mitral endocarditis. Since April there has

been great improvement with quinine and citrate of iron. The boy is stronger, less anæmic, and much brighter. The aortic murmur has been slowly disappearing, and now, although the aortic second sound is faint, a diastolic murmur can scarcely be detected even when the room is quiet. The pericardium is probably adherent. Features of this case: Marked improvement and coincident disappearance of the aortic murmur.

Case 5.—W. E—, aged 14 years. Severe attack of rheumatic fever at 11 years, lasting from November, 1902, to February, 1903. Symptoms—Tonsillitis, arthritis, nodules, aortic and mitral endocarditis, pericarditis; much cardiac dilatation. Under observation since that date. No further definite attack of acute rheumatism.

This case illustrates the combined lesion, which is compensated, although the aortic lesion is severe. The anæmia is marked, and all the aortic symptoms are quite definite. There is probably an adherent pericardium.

This boy goes to school and gets about well.

Since he has been under observation the two chief features have been: (1) the alteration of the mitral systolic murmur from a soft blowing bruit to a shrieking bruit; (2) an increase in the calibre of the pulse and a more distinct "water-hammer" character.

(*Exhibited May the 5th*, 1905.)

Discussion.

Dr. Theodore Fisher inquired what form of aortic disease was present in the cases which recovered. In some cases it was possible to mistake a very harsh pulmonary systolic murmur for an aortic one. He remembered seeing a case with an extremely good doctor who thought aortic stenosis was present, his own opinion being that it was a pulmonary murmur, and the subsequent post mortem proved that his view was the correct one. Did Dr. Poynton think it was possible in those cases in which the aortic disease disappeared that that condition might have been present? He thought all present would agree with Dr. Poynton's remarks on adherent pericardium. The important point in children's cases is the great enlargement of the heart

which is the rule in such cases. In nineteen cases out of twenty there was great enlargement where adherent pericardium due to rheumatism was present.

Dr. C. W. Chapman thought an important point in the prognosis was a large, strong heart, coupled with retention of the cardiac sounds.

Dr. G. A. Sutherland said that some years ago he would have said that an aortic diastolic murmur was a rare lesion in a child, but within the last seven or eight years he had met a great many more such cases than previously. He was surprised to hear that Dr. Poynton had met with so many cases, because he did not think aortic disease could be regarded as a very common lesion, more especially as regards the cases of regurgitation. He had never seen an autopsy in a fatal case. The fact of the lesion not being recognised during life would explain what he had said about the rarity of the cases, because possibly a number of them were overlooked, or, at any rate, the murmur was not easily heard. It seemed to him that if there was one clear distinctive murmur in connection with the heart, it was an aortic regurgitant murmur, both in adult life and in childhood. In going over his cases in the past few years he had never had any hesitation in diagnosing an aortic regurgitant murmur from its peculiar character. Dr. Poynton had stated that the aortic lesion disappeared occasionally. Did he mean that the aortic regurgitation murmur disappeared? Because he would say that was an unusual occurrence. After watching the cases for years, he had never seen one of them disappear, although at times there had been a temporary obscuration of the murmur. Dr. Poynton also said that a primary aortic disease by itself was rare. If all cases were followed up to the post mortem, one might be able to say that dogmatically, but he thought he had seen a considerable number of primary aortic cases in which there was no evidence of there being a lesion about the mitral valve. In regard to the clinical phenomena, he would like to ask Dr. Poynton if he had found as a rule that there was a well-marked spot at which the aortic diastolic bruit was best heard, namely, the third left intercostal space, close to the sternum. He thought the striking thing about aortic diastolic murmurs was how extremely well heard they were on the left side of the sternum; in fact, the part where they were not well heard was over the so-called aortic area.

Dr. F. J. Poynton, in reply, said that in each case there was an aortic regurgitant murmur and not a stenotic murmur. In considering the post mortems of such cases this disappearance of a murmur was quite conceivable, because one sometimes unexpectedly found at the examination the very slightest beading upon the aortic valve, although there had been no detectable sounds during life. It was also sometimes found at post mortems that the aortic valve was only so slightly thickened

that it was hardly likely to cause any aortic murmur. He thought what happened was that at first there had been sufficient beading to interfere with the action of the valve and give rise to regurgitant murmurs, and then, as it softened down and disappeared, a slightly thickened valve remained, giving no murmur. He had not been able to detect a murmur lately in the case he mentioned, although the patient used to have a well-marked one. He felt convinced that slight aortic regurgitation did disappear in children as did slight mitral regurgitation. The prognosis was exceedingly bad, because with poor children it was impossible to protect them from fresh rheumatism. In the present state of knowledge on the subject it was impossible to protect even the rich, and he was afraid the patient would not grow up to be 12 years of age. He agreed with Dr. Sutherland that the murmur was generally best heard to the left of the sternum. In the two boys between 13 and 14 years he had noted that the murmur had gradually shifted, as they grew up, from the left side across to the right—he supposed with the alteration in the position of the heart. The little boy, on the other hand, still had a well-marked murmur on the left side. He believed the mitral valve was almost invariably found damaged in the rheumatism of childhood when this was at all severe, and he could not bring himself to believe there was simple dilatation of the mitral valve in any of these cases, with very rare exceptions. In no post mortem on rheumatic children where aortic disease was existent had he ever seen the mitral valve not also damaged by actual disease.

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78. A CASE OF GENERALISED ALOPECIA.

By GEORGE PERNET.

Mr. George Pernet showed a case of generalised alopecia in a boy, aged 8 years, who had first come under observation about a year previously for bald patches (alopecia areata). The scalp was completely bald, and some attempt at growth had ended in shedding of the lanugo formation. The eyebrows and lashes had also been involved, but here there was improvement. The boy further exhibited the Mongolian (or at any rate Mongoloid) type of idiocy, with high

arch to palate, some uncertainty of gait, lateral nystagmus, and no doubt adenoids, judging from the open-mouthed aspect. There was no necessary connection between this general condition and the alopecia.

(*Exhibited May the 5th,* 1905.)

Discussion.

The Chairman (Dr. George Carpenter) said a similar case had been exhibited by Dr. Grünbaum at a previous meeting of the Society. Since then a boy, about 10 or 11 years of age, whose scalp was completely bald and shiny, had come under his care at the North-Eastern Hospital. He asked what treatment Mr. Pernet proposed to adopt, and whether cutaneous stimulants or drugs taken internally, or a combination of the two, proved beneficial in such a distressing condition.

Mr. Sydney Stephenson inquired whether alopecia affected the eyelashes. He remembered seeing one case of the kind.

Mr. Henry Skelding (Bedford) asked whether Mr. Pernet had used the X rays in such cases.

Mr. Ernest Clarke had seen a full growth of hair after several applications of the high-frequency current, the patient having been previously bald for years.

Mr. George Pernet, in reply to the questions which had been put to him, stated that in generalised alopecia it was not uncommon to find more or less loss of the eyelashes. As to the X rays, *à priori*, not much was to be expected from their application, seeing they were employed to remove hair. Mr. Pernet had not seen any result from high-frequency currents, although they might have given some result in the case mentioned. Stimulation and perseverance were the key-note of treatment.

79. A CASE OF DOUBLE COLOBOMA OF THE IRIS.

By ERNEST CLARKE, M.D.

Mr. Ernest Clarke showed a girl, aged 6 years, with coloboma of the iris, down and out, in each eye, associated with nystagmus. There was no other congenital defect.

(*Exhibited May the 5th,* 1905.)

80. BILATERAL PERIOSTITIS OF THE FEMORA, PROBABLY SYPHILITIC.

By EDMUND CAUTLEY, M.D.

Lilian L—, aged 12 months, came under observation two months previously for swelling of the lower half of the thigh and the knee on both sides. The right side had been affected for a month and the left for two weeks. She was the first child, born at full-time, of a tuberculous mother, and had been brought up on cow's milk and water for a month, and since then on Alison's food. She was small and wasted, weighing 10 lbs. 4 oz., anæmic, rachitic, and seemed ill. The two lower central incisor teeth were cut and the gums showed no discoloration. Her temperature was 103° F. The right knee was much swollen and the lower half of the femur was much thickened; the left was similarly affected to a less extent. There were considerable tenderness and pain on movement. Four days later the swelling on the right side was less, but that on the left had increased. Next day an incision was made down to the bone on the left side, a little above the knee, but no pus was found, only a little serum and blood coming away. After this the swelling gradually subsided and the temperature reached normal in two days. The progress was subsequently steadily favourable and uneventful, and the child was discharged four weeks after admission into hospital, having gained 26 oz. in weight, of a much better colour, and with merely thickening of the bones, which was still present. She had been given grey powder three times a day, and fed on milk, barley-water, Benger's food, and orange-juice.

The diagnosis of the affection was difficult. In favour of scurvy were the anæmia, the history of a diet peculiarly likely to induce scurvy, and the rapid recovery. Against it were the absence of any affection of the gums and the bilateral character of the swelling. Neither the pain nor tenderness were nearly so severe as is usual in infantile scurvy.

Acute osteomyelitis could be excluded, but the possibility of a mild form of osteomyelitis had to be considered. On the whole the affection was probably syphilitic, and an epiphysitis as well as periostitis. He based the diagnosis mainly on the symmetry, the course, and the absence of any other cause likely to produce a bilateral affection of this nature. The child showed no other indications of congenital syphilis, but the mother had had one miscarriage.

(*Exhibited May the 5th,* 1905.)

Discussion.

Mr. Henry Skelding (Bedford) referred to a case of a much older child, a boy, who had just been discharged from the hospital. He had been attending the Out-Patient Department with a thickening of the bones of the right forearm which was very painful and tender to the touch, without any improvement. The left forearm then became affected, and he was admitted into the hospital. Skiagraphs were taken of both forearms, and the right, which was first affected, showed thickening of the shaft of both bones. The left, although very painful, appeared quite normal, probably because calcium salts had not formed. With rest and arsenic the boy got completely well, although the thickening of both bones remained; the pain entirely ceased, and he was able to use both arms. There was no history of specific disease, and he was unable to say what the origin of the trouble was, but it was marvellous how quickly the pain disappeared after the administration of arsenic. In the Out-Patient Department he was given cod-liver oil and iron, but with little or no effect. One or two tender places on the long bones also quickly disappeared. It was interesting in such cases to take a skiagram. In the case he had mentioned the complete periostitis of the shaft was readily seen, and the joints were not affected.

Dr. F. J. Poynton said it was exceedingly difficult sometimes to diagnose these tender swellings. Not long ago he had the misfortune to see a child who had large swellings of the kind described in the femur, in which a confident diagnosis of scurvy was made. Antiscorbutic treatment was prescribed, and persisted in for some time, with no result. There followed the formation of large abscesses, with a considerable amount of damaged bone. There was no doubt that the child suffered from an unfortunate over-confidence in the diagnosis of scurvy. Only in the previous week he had an exceedingly puzzling case somewhat of the same nature in a child 1 year and 3 months

old, which had been fed much in the way described by Dr. Cautley. The patient had a large swelling in the femur and pneumonia in the right lung. There was no specific history, and no other signs of scurvy. Whether this was a case of acute periostitis or scurvy, or what it was, he was not prepared to say; it was exceedingly difficult to diagnose, and he personally thought it might be a pneumococcal periostitis.

Dr. G. A. Sutherland thought Dr. Cautley had dismissed the idea of scurvy very easily, without giving sufficient evidence, and accepted the diagnosis of syphilis without giving good grounds for the diagnosis. Such cases were extremely puzzling. At a children's hospital they were accustomed to seeing the three varieties of periostitis, namely the syphilitic, the scorbutic, and the acute septic. In the case under discussion Dr. Cautley found a certain degree of pyrexia. That was not very common in scurvy, but it was not unknown. Dr. Cautley gave an excellent history of the diet which would tend to induce scurvy, and also showed that the acute condition of the patient and the swellings in the femora subsided rapidly, presumably on account of the treatment in the hospital, which might be described as anti-scorbutic. On the other hand, in regard to syphilis, Dr. Cautley had produced no evidence whatever in the family history, or in the child's previous or present condition apart from the swelling. He might point out also that syphilitic epiphysitis at such an age was extremely rare. One saw a great number of such cases, but 99 per cent. of them occurred during the first six months of life, whereas in his opinion the child was too young for the later condition of syphilitic periostitis. He did not dispute the diagnosis, but he asked Dr. Cautley not to lightly dismiss the possibility of its being scurvy.

The Chairman (**Dr. George Carpenter**) said there was no doubt that swellings about the long-bones in children were sometimes exceedingly difficult to diagnose correctly. He called to mind the case of a child 10 months old, who was very anæmic and rickety and had a painful swelling about the right femur which he thought to be a case of scurvy. The child was treated antiscorbutically, but without result. An X-ray examination and a skiagram subsequently showed that there was considerable swelling of the periosteum of the femur, starting from the epiphysis and going halfway up the shaft of the bone. It remained quiescent for some time, but then gradually enlarged and finally suppurated. It was operated upon, and with a beneficial result. On the other hand, he had seen cases of undoubted scorbutic periostitis which had been operated upon by surgeons under the impression that they were suppurative. Neither extreme pain nor scorbutic gingivitis were absolutely essential to the diagnosis of infantile scurvy. He thought hæmaturia was a valuable sign of the condition—not necessarily

hæmaturia in quantity, but the presence of red blood corpuscles in the urine in small quantities requiring the centrifuge for their detection. He had found this mode of examination of use in difficult cases. Whether the recovery of Dr. Cautley's case was attributable to the grey powders or to the antiscorbutic dietary it was impossible to say. The combination was unfortunate in some respects. Dr. Sutherland had called attention to the fact that it was unusual to diagnose syphilitic epiphysitis at that age. He (Dr. Carpenter) had seen and recorded cases of syphilitic epiphysitis in such children, and he did not think its age put syphilis out of the question. The involvement of the epiphyses and the extension of the swellings up the shafts of the bones were, he thought, in favour of the diagnosis of syphilis rather than of scurvy, which did not select the epiphyses for attack.

Dr. Edmund Cautley, in reply to Dr. Sutherland, thought it was extremely rash to diagnose scurvy because a child got better on an antiscorbutic diet. In his own case, although the original diet was one on which scurvy was likely to be produced, there was absolutely no evidence of scurvy apart from the condition of the limbs. He had never yet seen scurvy sufficiently severe to produce swelling to the extent described, even in one limb, without some definite discoloration of the gums when the teeth were present. In his case, although there was considerable anæmia, the gums were not in the least discoloured. If Dr. Sutherland had seen the patient at the time, he thought he would have admitted there was no evidence of scurvy. It was a very just criticism to say there was little evidence of syphilis, but they all knew how difficult it was to obtain such evidence. It was by no means certain that the case was syphilitic. During the examination of the patient it had been pointed out to him that a rash on the buttocks was probably specific, but he did not think that was so. The grounds on which he laid the most stress of its being specific were first of all its chronic nature, secondly its symmetry, and thirdly that it had got well. Acute osteomyelitis could be excluded by the result of the incision, in the same manner that scurvy could be excluded, but he did not think one could definitely exclude a mild form of osteomyelitis, due to some micro-organism, analogous in that respect to the case Dr. Poynton referred to, one of periostitis in association with pneumonia. He thought it possible it might have been due to a bilateral mild form of osteomyelitis, starting in the epiphysis. Against a diagnosis of that nature was the fact that the temperature subsided very quickly three or four days after the child came into hospital, and remained down, and the fact that the disease was bilateral, and osteomyelitis was very rarely bilateral. One was, therefore, driven by a process of exclusion to a diagnosis of syphilis.

81. A CASE OF MUSCULAR ATROPHY, PROBABLY OF THE SO-CALLED "PERONEAL TYPE"

By F. PARKES WEBER, M.D.

The patient, Ethel B—, aged 11 years, was admitted under my care at the German Hospital on April the 26th, 1905, because of muscular wasting in the left leg and right hand. On examination of the left lower extremity there is found to be considerable atrophy of the peronei and of the whole calf and, to a much lesser degree, of the thigh muscles. The circumference of the left thigh, 5 inches above the patella, is $12\frac{1}{2}$ inches, that of the right thigh 13 inches. The circumference of the left leg, 4 inches below the patella, is $8\frac{1}{4}$ inches, that of the right leg $9\frac{1}{8}$ inches. The two lower extremities are of exactly equal length, as measured from great trochanter to external malleolus, showing that the growth of the bones in the affected extremity has not as yet been hindered. There is great wasting of the muscles of the right hand, especially of the thenar eminence, sufficient to produce a claw-like appearance, and the muscles of the right forearm are somewhat smaller than those of the left forearm. There is a certain amount of loss of power associated with the muscular atrophy, and the patient has felt a stiffness or difficulty of some sort in using her right hand for writing, but she can walk well and can dorsally flex her left foot like her right foot. There is a slight degree of "pes cavus" on the left side, as pointed out to me by my friends Dr. Arkwright and Dr. Cautley, but no pes equinus or equinovarus. The knee-jerks are present, but the jerk on the left side is less than on the right. No ankle clonus can be obtained. The plantar reflex on the right side is prompt and of the normal flexor type; that on the left side is sluggish and obtained with difficulty, but, when obtained, is likewise of the flexor type.

Sensation.—There is slight impairment for all forms of sensation (for touch, pain, and temperature) over the right

hand and lower part of the forearm, especially on the ulnar side of the hand, but sensation is good higher up the arm and in the left upper extremity. There is impairment for all forms of sensation over the left foot and, more markedly, over the outer side of the lower third of the leg. Sensation over the left thigh and the whole right lower extremity is natural, excepting in regard to electricity, as mentioned later on. No spontaneous pains are now complained of.

Electrical reactions.—Contraction to faradism is more sluggish and obtained with much greater difficulty in the muscles of the right hand (particularly the thenar eminence) than in those of the left hand. In the right thenar eminence the contraction to galvanism is very sluggish and diminished in amount, and ACC is greater than KCC ; in fact, there is a typical reaction of degeneration in these muscles. In the thenar eminence of the left hand the contraction to galvanism is brisk, and KCC is greater than ACC. Excitability to faradism is greatly diminished in the muscles of both legs, and no contraction is obtained in the muscles below the knees even when the faradic stimulus is strong enough to cause pain. The peroneal and calf-muscles of the left leg require a stronger galvanic current to induce contraction than do those of the right leg, but KCC is greater than ACC in both limbs. Sensation to faradism is much impaired in both legs.

With the exception of lessened irritability to faradism, which, as I have already mentioned, is very marked in the right leg, it appears that the only muscles as yet definitely affected in any part of the body are those of the left lower extremity and right upper extremity. There is no spinal curvature. The facial and oculo-motor muscles act quite naturally. There is no muscular inco-ordination to be observed in the movement of the limbs. No fibrillary twitchings have been noticed anywhere, neither have any unnatural vaso-motor phenomena. Ophthalmoscopic examination shows nothing abnormal.

The general nutrition of the patient is fairly good.

She is of medium height for her age, and weighs 3 st. 10 lb. She has slight chronic enlargement of the tonsils, but there is no fever or any physical sign of disease in the thoracic and abdominal viscera. The urine, of specific gravity 1025, is clear and free from albumin and sugar. The mental development is quite satisfactory.

History.—Her health has generally been good. The wasting in the left leg seems first to have been noticed by her father, about August, 1904, when she complained of some pain in the left knee, which lasted about a week. The father was likewise first to discover the wasting in her right hand; this he observed in December, 1904, when she told him that the hand felt stiff in writing. She has had measles, but this was as far back as 1901. No family history of a similar affection has been obtained. She is one of a family of six girls and a boy, all fairly well grown, though two of her sisters seem to have adenoid vegetations of the nasopharynx. Her father and mother are both living.

Remarks.—Syringomyelia is unlikely in the present case, for the characteristic sensory symptom of this disease (dissociation in impairment of sensation) is absent, and there is no lateral curvature of the spine. Owing to the insidious and practically painless onset and the distribution of the muscular atrophy, a pure neuritis is extremely improbable, and a diagnosis of anterior poliomyelitis would not account for the impairment of sensation, even were it to be allowed that chronic anterior poliomyelitis is probable in childhood. The most probable view is that the child, though the only one affected in her family, has commencing "muscular atrophy of the peroneal type," an affection first described, independently in the same year (1886), by Charcot and Marie in France and by H. H. Tooth in England. The atrophy is very asymmetrical at present, but it may become more symmetrical if the disease advances further.

"Muscular atrophy of the peroneal type" is by no means so limited to the peroneal muscles in its early

stages as this name by which the disease has been christened would lead one to suppose. Neither is the disease a pure primary amyotrophy (muscular dystrophy), as by some it has been supposed to be; and since both neuritis and degeneration of the posterior columns of the spinal cord have been found (by post-mortem examination) to be associated with the muscular atrophy in cases of this class, it is not surprising that sensory phenomena should constitute a part of the clinical symptom-complex. The decided reaction of degeneration to galvanic stimulation of the right thenar eminence is a noteworthy feature of the present case. Its presence, and likewise the presence of impairment of faradic irritability in the apparently unaffected (that is, not atrophied) right leg, strongly support the view that changes in the nerves constitute early, if not the earliest, lesions of the disease in question.

If the diagnosis of "peroneal type of muscular atrophy" be correct, there may after all be some connection between the measles in 1901 and the atrophy; for in many cases this form of atrophy has been known to follow an attack of measles or some other acute infectious disease. The attention of the parents was not directed to the wasting till the child chanced to complain of a pain in the knee in 1904, but in reality the wasting may have commenced much earlier. I have to thank my friends Dr. F. E. Batten and Dr. J. A. Arkwright for much information bearing on the subject of the present case.

(*Exhibited May the 5th*, 1905.)

Discussion.

Dr. Edmund Cautley thought the case was especially interesting in conjunction with one he showed at the Society three years ago.* There was no doubt it was what Dr. Weber had called it, a peroneal type of muscular atrophy. The "claw-like" condition of the hand was hardly ever seen in early life, except from this affection. He had no hesitation in stating that there was a certain amount of pes cavus, and he thought there was distinct atrophy of the vastus internus, both of which were

* 'Reports,' vol. ii, p. 163.

very characteristic features. The case he showed was very extraordinary in that it had started at an extremely early age. There had been definite indication of some deformity during the second year of life. As in Dr. Weber's case, apparently there was no history of congenital tendency. In a great many such cases there was a marked hereditary history, the disease being transmitted more commonly by the female than the male. Another peculiarity was that both patients had had measles. In his own case the baby had measles when it was 6 months old, and the symptoms of peroneal atrophy were noticed at $1\frac{1}{2}$ years, and it was quite possible they started earlier. He was inclined to accept the view that some of the isolated or sporadic cases were perhaps different in character and causation from the true hereditary type; and he thought one of the commonest causes was measles. He suggested to Dr. Weber that the disease in his patient had been started by measles. The toxin of the measles might have acted directly on the muscles, on the central nervous system, or on the peripheral nerves. Most probably the toxin had acted on the peripheral nerves, setting up a mild type of neuritis or primary nerve-degeneration. He thought that was a most likely explanation, and that, if the history of numerous cases were gone into, it would be found that in a certain number of them, especially where there was no hereditary tendency, there was a history of specific fever, most usually measles.

Dr. Parkes Weber, in reply, thought Dr. Cautley's suggestion in regard to measles certainly ought to be considered. He had himself suggested that possibly the disease might have begun slowly after the attack of measles. The child complaining of pain for a short time in one knee first drew the attention of the parents to the wasting in the leg, and subsequently when she complained of stiffness in the hand the muscular atrophy in the hand was noticed, but the wasting both in the leg and the hand might in reality have commenced long before it was discovered.

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82. A CASE OF CONGENITAL DEFORMITY OF THE HANDS: MANY MEMBERS OF THE FAMILY SHOW SIMILAR AND OTHER MALFORMATIONS OF THE HANDS.

By GEORGE CARPENTER, M.D.

The male infant exhibited, aged 3 months, shows truncation of both little fingers with absence of nails, and

Fig. 1.

Fig. 2.

Dr. George Carpenter's Case of Congenital Deformity of the Hands.

Adlard & Son, Impr.

Dr. George Carpenter's Case of Congenital Deformity of the Hands.

Adlard & Son, Impr.

a skiagram reveals absence of the terminal phalanges. The end of the ring finger of the right hand, which is shortened and but slightly longer than the little finger, is much broadened laterally.

The skiagram also shows a badly-formed middle phalanx, and the terminal phalanx has apparently toppled over and is affixed to the side of the digit. A small bone, the origin of which is not quite clear, is seen to one side of the middle phalanx (*vide* Plate I, fig. 1). The nail of the left ring toe is absent, and that of the right is rudimentary. The nails of both little toes are rudimentary. The infant, which is the youngest of six, is the only male child. The infant's two youngest sisters have truncated little fingers (*vide* Plate I, fig. 2, a skiagram of the left hand of one of these children; a skiagram of the hand of the other child showed a similar deformity).

The origin of the deformities appears to be in the mother's family on her father's side. The child's mother is the youngest of seven. Her little fingers are truncated and incurved, and there are no nails upon them. The skiagram shows rudimentary terminal phalanges (*vide* Plate II). The index fingers are the longest, and the metacarpal bones of the middle and ring fingers on each hand are shortened considerably, being but half the size of the index phalanges. The phalanges of these are correspondently long.

The mother's eldest sister and youngest brother have shortened incurved little fingers with no nails upon them. Of the eldest sister's children one has two nails on one finger and webbed fingers, and two are deficient in finger-nails. Two of the youngest brother's children have deformities of the little fingers of both hands. The mother's father had two thumbs on each hand, and the mother's uncle had webbed fingers of both hands. The skiagrams were taken by Mr. H. J. M. D. Sale-Barker.

(*Exhibited May the 5th,* 1905.)

Discussion.

Dr. C. W. Chapman stated that in 1900 he took two patients,

father and son, to the Clinical Society, whose condition was something like the cases described. All four thumbs were affected, and in the child there was entire absence of the radius in one arm. Skiagrams were published in the 'Clin. Soc. Trans.,' and he handed round the volume for inspection.

83. PHOTOGRAPH OF A CASE OF AMAUROTIC IDIOCY.

By GEORGE PERNET.

Mr. George Pernet showed a photograph of a male child, who was under Sir Victor Horsley's care at University Hospital in 1892, and upon whom Mr. Pernet had then done craniectomy several times for anterior microcephaly. The child was then (when photograph was taken) aged 1 year and 8 months. He was blind and had rigidity of the limbs, which were powerless. Mr. Pernet had brought forward the photograph to elicit the opinions of members as to the possibility of the case having been one of amaurotic idiocy, although he was aware that a retrospective diagnosis from a photograph was open to doubt. The eyes had not been properly examined. The child was Semitic in appearance, and so were the mother and the mother's sister, though not avowedly Jews. Mr. Pernet had lost sight of the child and did not know what had become of it, but he would try to trace the parents.

(*Exhibited May the 5th*, 1905.)

84. A CASE OF CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS.

By GEORGE CARPENTER, M.D.,

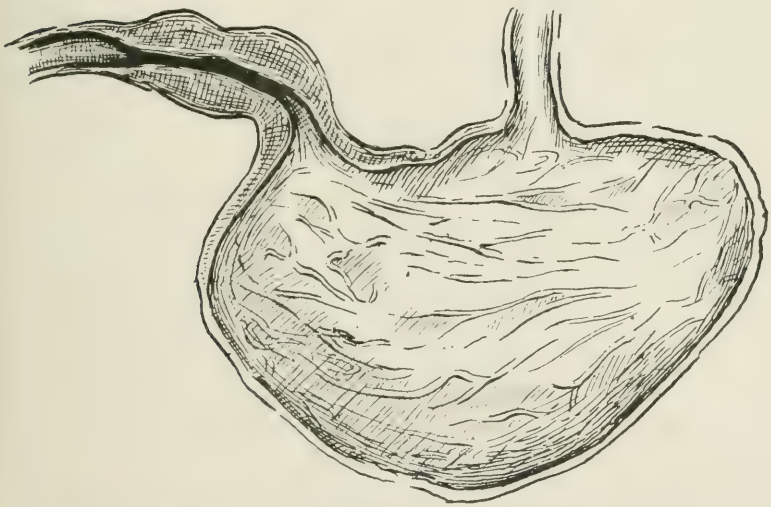
AND

P. LOCKHART MUMMERY, B.C.

The specimen shown was removed from a female child, aged 3 months. The infant was admitted into the North-

Eastern Hospital for Children, under Dr. George Carpenter's care, on March the 31st, 1905. It was born at term, the youngest of six living, and was breast-fed for the first two weeks, but nursing was discontinued owing to the formation of a mammary abscess. Vomiting sometimes occurred after suckling. The child was subsequently fed on milk and barley-water; the infant took the mixture

Fig. 14.



Longitudinal section of stomach and pylorus of a female child, aged 3 months—natural size—showing hypertrophy of the pylorus and of the adjoining muscular coat of the stomach.

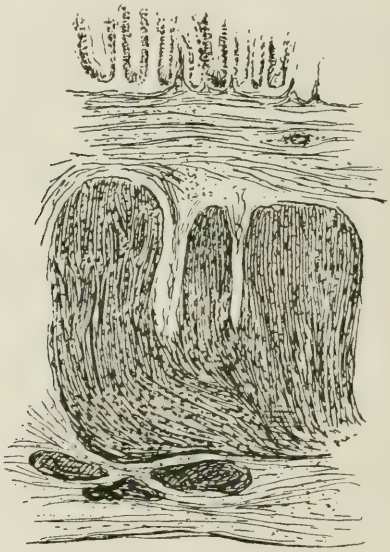
well, and vomited but little. It steadily wasted, however, and was always very constipated.

On admission it was an anæmic, wizen-faced, marasmic infant, weighing 5¼ lbs. It had a furred tongue and some thrush. The abdomen was flat and soft, and nothing abnormal was detected. The temperature was 99° F. On March the 31st it vomited twice, and on April the 2nd twice. On April the 3rd an hour-glass-shaped peristaltic contraction was seen for the first time in the epigastrium. The wave passed from left to right and then disappeared,

and at this time some regurgitation of the stomach contents into the mouth was noticed. On April the 5th the infant vomited three times, and on the 6th the peristalsis was again visible in the epigastrium, and it vomited once during that day, once on the 7th, twice on the 8th, but not on the 9th.

On the 10th it weighed 5 lbs., a loss of $\frac{1}{4}$ lb. since admission. About one inch above the umbilicus, and to

Fig. 15.



Longitudinal microscopical section of the thickened pylorus of female infant, aged 3 months.

the right of it, a hard cartilaginous mass, about the size of the terminal phalanx of the little finger, was felt lying deep in the abdominal cavity. The mass could be picked up between the finger and thumb through the abdominal wall. Manipulation of the abdomen caused regurgitation of food into the mouth, and simple flicking of the abdominal walls with the finger brought on visible peristalsis without hour-glass contraction. The stomach was dilated, its lower border being below the umbilicus, and the peri-

staltic waves passed from left to right and terminated at the right.

Dr. George Carpenter decided to have the infant operated upon, and Mr. P. Lockhart Mummery performed pyloroplasty on April the 11th. The infant was kept very lightly under chloroform, and an incision was made in the abdomen over the position of the pyloric swelling. A cut about $1\frac{1}{2}$ inches long was made through the pylorus, and this wound was then sewn up in the opposite direction over a small Mayo Robson's bone bobbin. The operation took about twenty-five minutes to complete. The child rallied well after the operation. Its pulse remained good during the night, and it took drachm doses of milk and barley-water half-hourly without returning them. It died, however, on the following morning. The post-mortem examination showed that a free opening had been established through the pylorus.

There were several points of interest about the case. The toughness of the hypertrophied portion of the pylorus was remarkable; the walls cut like the cartilage of the knee. It was also noticeable, on looking at the specimen, that the hypertrophy of the muscular coat extended for some considerable distance into the stomach.

(*Exhibited May the 5th*, 1905.)

Discussion.

Dr. Edmund Cautley thought the case illustrated two very important points. In the first place the operation should not be delayed until the diagnosis could be verified by definite palpation of the hypertrophied pylorus. The case showed how readily the tumour could be felt if the child was sufficiently wasted, but the wasting was a very deleterious condition. In the second place the prognosis was not the prognosis of the operation *per se*, but was the prognosis of the marantic condition at the time of the operation. He thought it would be found that a satisfactory result would very rarely be obtained if operation was postponed till the child was 3 months of age. These babies nearly always died before that age, whereas if the operation was performed within the first six weeks of life it might be said that practically every case would recover. He was surprised to find

so much difficulty experienced in doing pyloroplasty, having seen it performed at the same age with perfectly good results. One ought to be able to make the diagnosis very much earlier, and he thought the more quickly knowledge of the disease was disseminated the more often would the cases reach the surgeon at a reasonably early date. No matter what kind of operation was adopted, he was quite certain it ought to be carried out in the first six weeks of life and sometimes earlier. The cases he had had operated on early in life, about 1 month old, had all done extremely well. Pyloroplasty had been performed in all those cases, and he regarded it as the ideal operation, because he thought it provided the best remedy as a permanent cure. Loreta's operation in his opinion did not guarantee a perfect cure. Gastro-enterostomy produced a condition of the stomach which, if he were a child, he should not like to have for the rest of his life. He did not think surgeons were able to say with certainty that after a gastro-enterostomy the hole would not close up, or that the contracted condition of the pylorus would subside. In pyloroplasty the parts were very much tied up together, and for the next twenty-four or thirty-six hours, or possibly three days, extremely little food would be able to pass through the pylorus. The prognosis after the operation depended not so much upon the surgeon as upon the physician, because so much depended on the way the child was fed. Immediately after the operation patients should be fed with small quantities of food given by the mouth and by the rectum, but it was no use after pyloroplasty expecting food to pass through the pylorus at any rate for twenty-four or thirty-six hours.

Dr. F. J. Poynton said he had heard that if the cases were detected early operation was often avoided. Some physicians, he understood, hardly ever operated upon such cases, avoiding operation by washing out the stomach and careful feeding. He had seen one case—a case of Dr. Batten's—arrested with nasal feeding, the symptoms gradually melting away.

Dr. Cautley, continuing, remarked that he was strongly convinced that there were occasionally mild cases of the disease which might get well without operation, although he thought the washing out of the stomach had little or nothing to do with the result. He had seen a case of that nature, the specimen of which he subsequently showed at the Society. No operation was performed. The life-history and the post-mortem examination showed that during life food got through the pylorus in sufficient quantities for the child to live. Unfortunately, it got an attack of gastro-enteritis and died. He was perfectly convinced that 99 per cent. of the cases diagnosed as congenital hypertrophic stenosis of the pylorus and recovering without operation were not such cases. Because a child had a prolonged attack of vomiting one was not justified in making a diagnosis of that

nature. He believed in the existence of a spastic condition of the pylorus, without hypertrophy, which might subside under treatment, and he did not admit that such spasm would produce hypertrophy. He saw a case in consultation a short time ago with a view to operation being done, but he came to the conclusion that it was a mere spasmodic affection, and the child subsequently recovered without operation. He therefore thought that the condition could be diagnosed and cured without operation very frequently, provided one was easily satisfied with the diagnosis; but if the condition actually existed he believed it would steadily get worse and the patient would die as a rule under 3 months of age. There was definite evidence, however, that some patients lived longer, cases of 5 and 11 years of age and older having been recorded.

Dr. Theodore Fisher had seen it remarked that peristalsis of the stomach was not necessarily diagnostic of pyloric stenosis. In one case, which he sent in to hospital with a view to operation the peristalsis was well marked, but the child went out without an operation.

Dr. Cautley, continuing, said he did not regard peristalsis as an essential feature. He did not think the surgeon ought to wait for the presence of peristalsis or of a tumour before operating. Peristalsis might be present in other conditions, but he regarded it as a confirmatory symptom when the other symptoms were present. The most important indication was the history. When a baby, apparently healthy at birth, started vomiting at 2 or 3 weeks old, and that symptom subsequently became definitely characteristic, and when increasing constipation was associated with that condition, even on various diets in small quantities, if peristalsis occurred he regarded it as a confirmatory symptom. If the pylorus could also be felt, that was another confirmatory symptom; but he would advise operation without finding the peristalsis, and without feeling the pylorus.

Dr. Alexander Morison said in the case in which he showed a specimen some time ago the surgeon postponed operation as he was not convinced of the condition. The child was sent out of the hospital, ultimately came back in a collapsed condition, and died without having been operated upon. He felt strongly with Dr. Cautley from his own experience that the pyloroplastic operation was the correct one, especially compared with that of stretching the pylorus. Some of the cases thus treated undoubtedly recovered, but he was perfectly convinced that one of the cases he had seen treated by stretching had died in consequence of an œdematous condition arising from the laceration and bruising which absolutely occluded the pyloric orifice.

The Chairman (**Dr. George Carpenter**), in reply, remarked that Dr. Cautley had called attention to the necessity for early

diagnosis. Early diagnosis did not take place in his case, for the reason that he did not see the patient until it was 3 months old; the condition was diagnosed immediately he saw the child. He was in entire agreement that cases should be operated on early and before marasmus had set in, because it was well recognised that when infants had reached a marked marasmic state it was impossible to save them; the prognosis, therefore, did not depend upon the condition of the pylorus, but upon the condition of the body. But before early operation could occur the patient had to be seen. He called attention to the fact that gastric dilatation was no necessary part of the complaint—there might be pronounced pyloric thickening with an undersized viscus. In such cases visible peristalsis was absent. He was of opinion that the condition of the pylorus could always be gauged by examining the infant in the proper way for its detection; and the proper way was, not by palpation, which was a very unreliable method, but by methodically squeezing all the parts underlying the abdominal muscles between the fingers and thumb. The hypertrophied pylorus was very elusive; it was apt to fall right away from the abdominal parietes, deep down into the abdominal cavity, and it could be readily missed even when large and under an anæsthetic, and he had seen the condition overlooked by skilled examiners who relied upon the older method of examination. He was not quite in agreement with the statement that symptoms apart from physical signs were sufficient to mark the disease. Unfortunately, symptoms were not always present to help the physician, the history may be indefinite and certainly unconvincing, and even when the case comes under observation vomiting may occur only occasionally. It was so in the case he had just narrated, and yet there was a considerable pyloric tumour in that patient, and he on the evidence and symptoms would certainly not have considered it a case of pyloric stenosis had it not been for the results he had obtained by a physical examination. The absence of classical symptoms had not been an isolated experience—he had met with it before. In the presence of symptoms of obstruction associated with dilatation of the stomach and peristalsis he would hesitate to advise an operation before making a very careful abdominal examination in the manner he had indicated. Failing to find thickening of the pylorus—and if it were present it ought to be felt—he would try medical measures and would not resort to an abdominal section. Not only could pyloric enlargement be readily felt by his method of examination, but the thickening of the adjoining stomach walls which occurred in this case, and which he had also observed in another example, was easily detected, a sufficient tribute to its delicacy and reliability. It was early days to speak with great confidence as to the best form of operation—the cases on record were not sufficiently numerous to dogmatise.

He had seen Loreta's operation quite successful from a mechanical point of view, though the infant did not eventually recover. He had also seen an example where pyloroplasty would have been impossible owing to extensive pyloric infiltration of cartilaginous hardness quite unadaptable to such a procedure.

Mr. Lockhart Mummery, in reply, said he did not mean to say that pyloroplasty was bad because it was a difficult operation, because he did not think there was very much difference in the difficulty between the two operations. The important factor in a case where the child was marasmic was the speed with which the operation could be performed. A child might stand the operation fairly well, provided it was in a well-nourished condition and provided the operation was not long; but in the case under consideration the child was in a bad condition, and he thought as gastro-enterostomy could be done in about half the time it was the better operation to perform. It did not seem to be a reasonable operation to cut through tissue nearly as tough as the cartilage of the knee and sew up the incision in the other direction as in pyloroplasty, because, owing to the thickness and hardness of the tissue, so much kinking was caused that a free passage through the pylorus was difficult to obtain.

85. MULTIPLE GUMMATA.

By J. PORTER PARKINSON, M.D.

Florence B—, aged 7 years, the ninth of ten children mother had three miscarriages at various times.

This child was born at full-term, and has never suffered from any skin-eruption either as an infant or since.

She had a small abscess opened in the forearm on April the 7th, and now she has the following lesions: various softening areas implicating the skin on the outer side of each thigh; one or two others deeper; a similar one on the inner side of the right leg just below the knee, involving the gastrocnemius and soleus muscles; thickening involving the tendons at the radial side of the right wrist.

There are two small lumps on the shins.

She has not the aspect of congenital syphilis, and there are no signs in the viscera. The incisor teeth are pegged and separated from each other.

(*Exhibited May the 5th*, 1905.)

86. A CASE OF BIRTH PARALYSIS.

By J. PORTER PARKINSON, M.D.

This child, aged 4 months, was a firstborn. The labour was very protracted, lasting over twenty-four hours, and the mother was told that it was a "cross birth" and that the arms came first. Since birth the arms have been useless, and though not completely paralysed, are somewhat rigid and contracted. The electrical reactions taken by Mr. Sale Barker show that the lesion is one of the lower motor neurons, probably in the brachial plexus, and there is no evidence of intra-cranial hæmorrhage, the more usual cause of these paralyses. On the left side there is complete reaction of degeneration in the muscles of the forearm and hand. On the right side there is a fairly good reaction to Faradism and K. C. C. > A. C. C., but the contraction is somewhat sluggish.

(*Exhibited May the 5th,* 1905.)

87. A CASE OF LARYNGEAL PAPILLOMATA—DEATH FROM ASPHYXIA.

By LEWIS MARSHALL, M.D.

Fred H—, aged $4\frac{1}{12}$th years, was admitted into the Nottingham Children's Hospital on April the 25th, 1905, under the care of Dr. Lewis Marshall, suffering from "difficulty in breathing."

History.—The patient had always been strong, and had not been attacked by any infectious disease. Two years ago his voice was gradually lost, until he could not speak above a whisper. This condition had continued ever since. Seven months ago breathing became very difficult, and diphtheria was diagnosed, and tracheotomy was performed at the Fever Hospital. "When the trachea was opened a quantity of pus escaped, and no diphtheria was found."

The tube was removed after seven weeks; breathing was not embarrassed, and the patient was able to whisper as before. For three months before admission into the Children's Hospital the parents had noticed "sucking-in of the chest and neck at each breath." On the night before admission the patient had an attack of breathlessness, "turned blue, and fought for breath." (The father refused permission for tracheotomy should it become necessary.)

On admission.—The patient looked healthy. He was a good colour. He could not speak above a whisper. The chest was pigeon-breasted, the veins not enlarged. There was marked inspiratory recession of the muscles of the neck, the lower intercostal spaces, and the diaphragm. There was neither cough nor expectoration. No enlarged glands were visible. The chest was resonant all over. Air-entry was deficient in both lungs, especially the left. Forty-eight hours after admission the patient died, after a period of three hours' increasing cyanosis and unconsciousness.

A post-mortem examination was made, and the larynx was found to be almost occluded by warty growths, which extended on to the epiglottis.

At the bifurcation of the trachea was a mass of enlarged glands, lying over and compressing the left bronchus.

The anterior borders of both lungs were emphysematous, and hæmorrhages were seen on the surface of the left lung.

(*Exhibited May the 5th,* 1905.)

88. THE WIGHTMAN LECTURE: SOME OF THE NEUROSES OF EARLY LIFE.

By HENRY ASHBY, M.D.

Mr. President and Gentlemen,—In these days of lectures and addresses, to say nothing of the intense activity displayed in all departments of medicine, it is not easy for a

lecturer to find an untrodden path or feel that he can add anything to the common stock of knowledge. I must crave your indulgence if—in delivering the address which your Council in its goodness has imposed on me—I traverse some rather open and well-known ground.

Important contributions to our knowledge of the neuroses of early life have been made in this country by such well-known observers as Gee, Warner, Shuttleworth, John Thomson, Still, and many others; while in the allied domain of social psychology the names of Romanes, Sully, and Mark Baldwin will at once suggest themselves. To all these writers I must express my deep indebtedness. It is not easy to define the limits of a neurosis, but the task which I have set before myself to-day is to speak of the functional disorders due to a failure of co-ordination or a backward development of the lower nerve centres, and of the minor mental abnormalities occurring during early life due to a lack of inhibition or a morbid sensitiveness of the higher level centres. It need hardly be said that I am not attempting to cover the whole ground occupied by the neuroses and psychoses of early life, for to sum up all the moods and tempers of the twentieth-century child would require a volume. I must be content to pass in review only a few, and perhaps be deemed guilty of omitting some of the most important.

There is some difficulty attending the use of the term "functional disorder"; what one may call a "functional disorder" or "neurosis," another with good cause may say "What you call a 'neurosis' is only an exaggeration of what is seen in every child when over-excited, or tired, or in ill health." Or, on the other hand, a neurosis such as eclampsia may be due to some organic change in the nerve cells or some focal lesion. I must risk such criticism, bearing in mind that there are no hard lines in the classification of disease and there is great comfort in taking refuge in the word "borderland."

Since Darwin, "variation" has been looked upon as one of the broadest facts of biology; differences of disposition,

temperament, and character are met with in members of every family. It is, however, I think, a matter of common experience that neurotic parents have neurotic children. Heredity does not necessarily show itself in fully-developed disease at birth or in every member of a family; an imbecile woman does not necessarily bring forth an acephalic fœtus or an epileptic or insane father an imbecile. There is, however, a strong probability that if a family results from the union of neurotics or degenerates some members of that family will show a tendency to unstable nerve centres or mental feebleness. There is no strain through the generations so strong and persistent as the tendency to unstable nerves and mental degeneration. In nature the weakly and unstable are weeded out before they have the chance of breeding. In our highly organised and philanthropic civilisation the weaklings and neurotics are tided over their infancy by thousands and preserved till manhood. Social conditions rather than natural laws regulate the union of the sexes.

Are we more neurotic than our forefathers who followed the tail of the plough and lived upon the produce of their land? Have the unrest and excitement of these later days told, not only upon men and women, but also on their offspring? In our large cities we are beginning to discover a large accumulation of "by-products" or "*Nebenconsequenzen*" in the shape of the criminal, the pauper, the epileptic, the imbecile, and the insane, with the prospect at no distant date, as several writers have pointed out, of one half of the population having to nurse, to cherish, and to support the other half. The individual born to-day repeats the gains of former generations, the individual gains becoming race gains; but is it not equally true that in the long run individual degeneration becomes race degeneration? The fertilised ovum has implanted on it, not only the characteristics of races, families, and individuals, but it has been modified by climate, nutrition, and toxins. It is "the heir of all the ages in the foremost files of time." Well did the Egyptian write over the Temple of Isis: "I

am whatsoever has been, whatsoever is, and whatsoever shall be, and the veil on my face no mortal hand has ever raised." In addition to inherited tendencies there is the influence of nutrition and environment during the period of intra-uterine life. During the period when nerve and nerve cell are being shaped out of germinal plasm the progress of development cannot be indifferent to surroundings.

What are the essential features or characteristics of a neurosis or psychosis in a child? In attempting to answer this question we must bear in mind both age and state of health. What may be normal in a child 2 or 3 years of age may be very abnormal in a child 10 years of age. Then ill-health, over-indulgence, and bad home influence are very apt to bring out the unlovable and "seamy" side of child life. Among the characteristics we may note: (1) Want of co-ordination in muscular movements, both reflex and voluntary. (2) Hyperæsthesia of motor and sensory centres; a slight stimulus provokes a strong reaction out of all proportion to its strength. (3) Failure of inhibitory control over actions or reflex movements. (4) Exaggerated conscientiousness and contrariness. (5) Certain morbid habits not seen in normal children. (6) Tendency to *petit mal* and hysterical attacks. That there is overlapping in these characters is certain, but overlapping is not easily avoided.

In coming to close quarters with the neuroses of early life let me first of all refer to some of those that are very early in evidence and which, perhaps, depend upon developmental causes. The respiratory centres and also the deglutition centre must be in a condition of functional activity if life is to be maintained, but at times we find that the muscular movements which depend upon these centres are not perfectly performed. The movements depend upon muscle stimulations and inhibitions, which must take place in orderly and regular manner: there must be perfect timing or else there is a hitch. Presumably the movements are controlled by a superintending co-ordinating centre which is in charge. In congenital stridor

there is apparently a failure of inhibition or an active stimulation of the adductors of the cords and the expiratory muscles during inspiration, which is most in evidence during excitement or crying. The cords are more or less approximated during inspiration and stridor results. As the infant grows older the stridulous sound is less and, perhaps, is only present when deep inspirations are taken as in crying. As a rule infants who are thus affected are perfectly healthy and normal in other ways, and the stridor disappears during the second year or before. Infantile stridor is, however, in some cases associated with other defects. In one of my cases the infant had very marked stridor and also a posterior meningocele. Death occurred at 5 weeks of age during a severe spasm. At the post-mortem examination the naso-pharynx and larynx were perfectly normal. In another case stridor was noticed at birth, and later it became clear that the infant had also cerebral diplegia and mental defect. The child had a habit of letting off superfluous energy when excited by forcible inspirations and expirations accompanied by loud stridor. She is now aged 12 years, and still has the same habit of forced respirations when she meets a stranger, but years ago the stridor ceased. In another case, that of a boy, aged 10 months, there is marked stridor at times which has been present from birth; this infant has an older brother who suffers from diplegia and athetosis. In both these cases of diplegia I believe the cause was developmental and not due to a lesion at birth. It is no doubt true that in some cases nasal obstruction may give rise to stridor or snoring as suggested by Dr. Eustace Smith, but I think, and indeed there is post-mortem evidence to show, that the association is not a necessary one. Excitement, and very likely also bronchial and laryngeal catarrh, accentuate the stridor.

Deglutition is sometimes performed with difficulty in newly-born infants and in young children. Any nasal obstruction—such, for instance, as is present in congenital syphilis—will render swallowing difficult, but in other cases

there is nothing to explain the difficulty except a failure of co-ordination. It is not always well marked in breast-fed infants as they cannot draw much into the mouth at a time, but mothers will notice the delay in swallowing. The difficulty is most marked when bottle-fed and when the infant is in a great hurry for his food. There are choking and spluttering unless the act of swallowing is performed slowly and with great deliberation. The fluid is "gargled" in the throat instead of being swallowed, but there is no necessary return of fluids through the nose as in paresis of the soft palate. Feeding with a bottle is often so troublesome that a teaspoon is resorted to, and the mother very soon gives the infant sops. One girl I have watched is now 3 years old and still has much difficulty with fluids, especially when thirsty and in a hurry to quench her thirst. She has no difficulty with sops, but can only get down a few tablespoonfuls of fluid in the day.

The readiness and ease with which infants vomit are well known, the vomiting reflex being always on the *qui vive* and easily excited, but in some infants this is very marked. After making every allowance for overfeeding and improper food, a certain class remains in which this constant vomiting is due to an over-sensitive or uncontrolled action of the vomiting centre, and we can hardly do otherwise than look upon this as a neurosis. Nocturnal bed-wetting in children who have long passed babyhood, and who have never managed to pass a night in their lives without micturating during sleep strongly suggests a developmental failure of the controlling nerve centres which co-ordinate micturition. There is an uncontrolled action of the urine-expelling mechanism, or a failure to inhibit.

Head-shaking and nodding are also co-ordination neuroses associated with purposive or instinctive movements which become confirmed into a habit. Attempts to hold up the head and to turn it from side to side or up and down in order to obtain a better view of surrounding objects begin in normal children about the third or fourth month of life. These head movements are necessarily associated with

movements of the eyes. They are not voluntary movements in the strict sense, as they are not the result of a clearly pictured idea or revived memory image, but are rather "suggestion reflexes"—*i. e.* reflexes which come through consciousness.* These head-shaking movements may be noted as early as the third or fourth month with the first attempts to hold up the head and to look about, but the infant may not be brought for medical advice for some months after this. Instead of the muscles which move the head and eyes being stimulated and inhibited in a regular and orderly manner the action of the nerve centres is intermittent and produces, in addition to the purposive movements, constant jerky ones. The tremor becomes more marked, especially the nystagmus, when the infant is intently gazing at some attractive object. Probably habit plays a prominent part in confirming the movements. As time goes on and the infant gets into better health and exercises more and more voluntary control the movements gradually cease. These movements are chiefly seen in infants and children of low vitality, and many of them are rickety in more or less degree, but I have not seen these movements in cases of severe rickets. Sometimes they occur for the first time after an illness, such as pneumonia, and in one case related to me nystagmus came on for the first time after an operation for the cure of a hernia. Apparently the stammering, which is likely to come on during the second or third year is essentially very much on a par with head shaking and is apt to become confirmed by habit.

Another group of neuroses of the lower neurons includes such diseases as spasmodic asthma, recurring fever, recurring vomiting, and perhaps diabetes. All of these are especially apt to occur in neurotic families and give evidence of their presence early in life. The type of spasmodic asthma seen during infancy is usually Henoch's gastric type, but asthma secondary to a bronchial catarrh or nasal

* See 'Mental Development in the Child and the Race,' Mark Baldwin, p. 104.

irritation is also seen. A typical case of asthma in an infant was shown at this Society a year or two ago by Dr. W. Ewart, and I have recently seen one (with Dr. A. A. Mumford) in which the asthmatic attacks began at the age of 8 weeks. The exciting cause appeared to be difficulty of digestion, beginning with weaning. The father had suffered from spasmodic asthma as a boy, and the infant's mother's brother was also an asthmatic. Between the attacks the infant was quite comfortable, while during the attacks the chest was markedly drawn in at the bases and bulged out at the apices, and there was great distress from the dyspnœa. There was no evidence of enlarged thymus. In another case I saw during infancy distension of the stomach with gas preceded the attack, which was relieved when the distension passed away with the dispersion of the gas. This patient is now over 20 years of age, and has suffered from attacks of asthma all his life. The attacks seem to be excited by some irritation of the terminations of the vagus nerve.

Recurring fever also is apt to occur in members of a neurotic family and to commence in early life. It is well known that in neurotic children the temperature goes up from slight causes or without any apparent cause. In some cases, as is well known, these fever attacks recur again and again at intervals of a few months without being associated with any common or uncommon complaint. The boy's temperature runs up to 104° or 105° F. without any sign or symptom in the chest or elsewhere, he seems fairly comfortable and his appetite is good, the fever continues for some days or a week or more and disappears, leaving him weak and pale for awhile. No doubt in some cases the exciting causes are over-feeding and an accumulation of by-products. These cases are certainly apt to occur in neurotic families, and perhaps also tuberculous families, and it is important to note that Dr. G. H. Savage has noticed some cases of mental breakdown in young adults who have suffered from these recurring attacks of high fever.

Let us pass now to some of the minor mental abnormalities of early life which are associated chiefly with the higher level centres. It is not difficult to recognise very early in life that some infants are much more easily aroused than others, and are far more sensitive to impressions. All babies cry when they are hungry or have internal discomforts, and, speaking generally, the crying reflex is in good order during infancy, but some infants utilise it much more than others.

We all know the neurotic infant: he is a bad sleeper, he is awake half the night, he wants a lot of rocking or walking about; the slightest noise or discomfort awakes him. He is very apt in consequence to be over-fed, as he has no compunction in making his wants known, and gaseous distension of the abdomen and colic aggravate his normally hyperæsthetic disposition. He is early subject to fear, has an exaggerated dread of falling, and starts and clings to his nurse if swayed about. Many of these infants, as I have already said, are troublesome vomiters. Excitement of any sort may start the vomiting reflex. One small girl I knew used to suffer from nausea when the bells of a church close by began to ring for evening service. When about 2 years old she used to say "Church bells make baby sick," and at times she not only had nausea, but vomited. In later years she suffered from attacks of migraine and hysterical half-blindness. She could read the first half of a two-syllable word, but not the last half, while the headache was present. The same class of child shows, perhaps, excessive timidity all through infancy and childhood. Every sudden sound makes them start, whether it is the rattle of a spoon or the barking of a dog. At the sight of a stranger or an animal they will turn white and cover up their faces. They have a perfect horror of being stared at and find refuge in a prolonged cry. They dread the sight of the medical attendant, who probably reciprocates their feelings. As the child becomes older the timidity becomes a perfect nightmare. They are always seeing uncanny things, and have an unwavering belief in

ghosts and bogies. R. L. Stevenson must have been thinking of his own childhood when he wrote :

> "The shadow of the balusters, the shadow of the lamp,
> The shadow of the child that goes to bed,
> All the wicked shadows coming tramp, tramp, tramp,
> With the black night overhead."

If daylight relieves them of their fears, they return with unfailing regularity with the evening. When they go to bed the room must be lighted and the door open till confidence is in part restored by the nurse coming to bed.

It is for the most part the same class of child who has dreams of the tragic sort. The child during the day has been frightened by dogs, or stories about rats, or has made the acquaintance of strange beasts in picture-books. A marked impression has been made on the visual centres and the images come back during sleep, associated with the curtains or surroundings of the rooms. In rarer cases the hallucinations occur during the day and in the waking hours, or in a well-lighted room at night before going to sleep. The child sees animals under the bed or table, and will point them out to her mother, absolutely believing in their existence. Or she will call up her mother at night and describe ducks which she has seen in the park in the morning waddling about on the walls of the room.

In some children there is, perhaps, not only hypersensitiveness, which shows itself in bitter tears at trifling causes, but a contrariness which makes them difficult to manage. There comes a period in many children's lives when they develop a certain marked determination to disobedience, and take apparently the greatest pleasure in doing the opposite to instructions. In some neurotic children this phase becomes almost unbearable. They are spoilt by their parents, who laugh at their odd ways or scold them for the same thing next day, but they generally manage to get the upper hand and to rule the household with a rod of iron.

In another class of child we find a want of control, not so much over tempers, but over desires, and a failure in the

growth of an educated conscience. The child emerging from infancy wants every toy he sees, regardless of the rights of others. But as intelligence dawns he gradually shapes his actions in harmony with his surroundings and conforms to the unwritten laws of the society in which he is reared. The child with feeble powers of control gratifies his desires without any regard to others and without any inward sense of shame and will purloin toys, money, jewellery, anything that attracts, often in a clever way. Such children are often bright and clever and without a stain on their characters, until it is found out that they are habitually stealing from their playmates. They will confess up to just so much as they are obliged, and will explain that they cannot help it. In the worst and confirmed class of case they are moral imbeciles and amongst the worst of life's failures.

In yet another class, instead of a want of controlling power, there is an excessive inhibition over action and desire. These children are too good and very sensitive to reproof. They have a morbid fear of being late at school and lie awake half the night from anxiety lest they should fail to know their lessons next day. They get, perhaps, more morbid as time goes on, accuse themselves of faults never committed, surreptitiously take off marks given them at school by their teachers, are anxious to be punished for sins never committed, or to sacrifice themselves for the good of others.

What is likely to be the future of these erratic and abnormal children? Much depends, no doubt, upon heredity, but who can doubt the influence on their career of environment during early life? It is certain that an over-sensitive child will ill withstand the world's buffetings, and has more offended feelings in store than fall to the lot of ordinary mortals. But assuming they are carefully tended and there is no evidence of *petit mal* or anything morbid, the future is hopeful enough. There is, however, always a fear that a breakdown may come after puberty or during the strain of early manhood or womanhood. In

this class we are likely to find hysteria, migraine, and insanity.

The contrary boy is, on the whole, a promising boy if placed under reasonable control. As heretic and reformer, he has played his part in the world's history. Some of the ablest and most successful men I know, whose only fault is a weakness for argument, I feel sure once walked through all the puddles on the highway and defied their nurse at bedtime. In those who exhibit a want of control over their desires we have the making of criminals—or shall we say successful business men?—while those with over-sensitive and exaggerated self-control may show in later years the highest forms of self-abnegation, or if they lose their mental balance, are only likely to pass through religious excitement to hopeless insanity.

While an over-sensitive nervous system during early life may be accepted as a warning signal, a condition of the senses and feelings which can be described as morbid is at least suggestive of something worse. Among these morbid phenomena may be mentioned the masturbation habit and the dirt-eating habit, while a tendency to eclampsia, marked destructiveness, impulses to scratch and bite, and cruelty to animals may also, perhaps, be included among morbid habits. All through infancy movements, whether simple reflexes or "suggestion" reflexes in which the stimulation comes through consciousness, tend to repeat themselves when accompanied by a pleasurable sensation and a habit becomes established.

The masturbation habit is not uncommon both in boys and girls as early as the sixth or seventh month of life. Apparently it takes its origin in a hyperæsthetic state of the pudendal nerves; certain instinctive movements follow which give satisfaction and a habit becomes formed. This is seemingly on a par with the teat-sucking or thumb-sucking habit. When hungry or in discomfort the thumb or dummy teat finds its way to the mouth, gives satisfaction, and distracts from the discomfort; this satisfaction is intensified by the sucking movement and a habit is formed which

goes on day or night. While both habits are bad, it is certain the masturbation habit is doubly so, and points to a perverted state of the nervous system. The habit appears more persistent in boys than in girls, but this may be, as Dr. J. Thomson has suggested to me, that mothers and nurses discover the habit in boys but not in girls. The infant will cross her legs, work them vigorously, with flexion and extension of the hips, or sit in a chair swaying to and fro till almost exhausted and the perspiration breaks out. Similar movements may go on at night when half asleep. The habit can be broken by a good nurse with a strong personality and persistence; careful attention to health is, of course, necessary. Circumcision may be necessary in boys, though in one case coming under notice masturbation began for the first time shortly after circumcision. Too much need not be made of this habit, especially during infancy, nor need we attempt to judge the morality of the infant by the standard of the older child or adult. He is, after all, only the "raw material of morality." *

The dirt-eating habit is another instance of a perverted morbid taste which passes into a habit and may be seen during the first year or later. In its lighter forms it is not uncommon but always uncanny. Perhaps the commonest form of pica consists in picking blankets and swallowing the wool or gnawing clothes; this, perhaps, is associated with strange tastes as regards food and a liking for nauseous medicine. As soon as the child has learned to crawl the range of his depredations becomes more extended and various queer objects are requisitioned for dietetic purposes: plaster from the walls, cinders, bits of paper, slate pencils, with perhaps a special predilection for licking the fender and the blacking off boots. Taken by itself, perhaps, dirt-eating does not mean much, but as it is so frequently associated with imbecility and insanity, it suggests an abnormal condition of the nervous system. In otherwise normal children the habit is gradually broken as time goes on. No doubt the tendency to imitate which all healthy

* See 'Studies of Childhood,' Sully, p. 229.

children acquire and a feeling of shame at practising baby ways help in the way of reform. If, however, as the child grows older he continues to show morbid tastes as regards queer things, and if this is associated with other abnormal signs or *petit mal*, the future is not so hopeful. Many of these dirt-eating children are sallow and unhealthy-looking, suffer from diarrhœa, and all sorts of articles which they have swallowed make their appearance in their stools.

Another habit, more or less morbid, which develops at times in neurotic infants is a trick of suddenly digging their nails into the hands or face of their nurse or anyone who picks them up. It comes too early in life to be described as viciousness; it is rather an impulse which becomes a habit, the child deriving pleasure from noticing the consternation it produces. In most cases the habit is gradually broken as the child grows older. On the other hand, children who are more or less mentally abnormal or weak retain some such habits for years. They may be really dangerous in their ways and quite unfit to play with other children and get a bad character as playmates. The same heedlessness may be shown in the treatment of dogs and other domestic animals; they seem devoid of sympathy and heedless of pain. Such children are given to the masturbation habit and not infrequently suffer from *petit mal*. Their future is none too promising.

Time will not permit of any long reference to the various forms of major and minor epilepsy which occur in children. During early life we are constantly reminded of the close relation which exists between infantile convulsions, hysteria, and the various forms of epilepsy. We see in children forms of slight fits in which there is no complete loss of consciousness, a momentary "faint," a stumbling when at play, or some co-ordinated movement, such as making grimaces, getting up and sitting down again, which appear at first to the parents as being only "tricks." They might pass for such were there not a temporary pallor, a dazed look, or perhaps at times a real and undoubted convulsion. These minor fits are often more or less under the control of

the individual; a sharp word or a shake will ward them off. They tend to grow worse if much notice is paid to them and the child thinks that her fits are a source of interest to the family circle. They tend to cure if treated with wholesome neglect, a scolding, and cold water. There is always the fear that they may be really epileptic or drift into a condition in which epilepsy alternates with hysteria. Such attacks are closely allied to epilepsy and occur in families in which some member is undoubtedly epileptic, but differ in their type from typical epilepsy, inasmuch as there is no complete loss of consciousness, the movements are purposive or co-ordinated, and they are more under the control of the will.

I am inclined to think that such attacks as I have described tend to get well, and the prognosis is much better than in typical epileptic seizures. Let me make perfectly clear that the bad habits and bad ways which I have described, if occurring in otherwise normal children, need not give rise to any great anxiety if they are accepted as warning signals. If, however, they persist far longer than the period of infancy, or when there is a combination of morbid habits or there is any sign of mental feebleness, the future must be regarded as uncertain. Epileptic seizures, however slight, if associated with destructiveness or mental backwardness, are anything but reassuring symptoms as regards the future.

I have already referred to the fact that during intra-uterine life both heredity and environment help to give shape to the human organism. During infancy we have hereditary influences still in the ascendant, but in addition to these we have the added influence of personality and training. The brain trebles in weight during the first year of life and continues to increase with the body weight, and who can doubt that as the nerve centres are being perfected and the nerve paths opened up the influences brought to bear on the individual will materially help to mould the character and shape the future life. "Very early in life," to quote Baldwin, "the infant responds to suggestions of

personality. As early as the second month it distinguishes its mother's or nurse's touch in the dark. It learns characteristic methods of holding, taking up, patting, and adapts itself to the personal varieties. This is quite a different thing from the child's behaviour towards things which are not persons. I think this is the child's first step towards a sense of the qualities which distinguish persons."* The nurse puts the infant off to sleep when no one else can, soothes and quiets him when in discomfort, and appeases him when in fractious mood. During the first few months, when the infant is still in the instinctive stage, the formation of habit begins. In the dim consciousness of these early days he is a creature of habit. The infant easily falls into serviceable or bad or morbid habits. Pleasures and pains lead to motor discharges or instinctive movements, and movements once started tend to become repeated or stereotyped.

It is clear beyond doubt that the personality of the mother or nurse counts for much during the earliest months; the intelligent nurse can form good habits in feeding, care, and hygiene, and break up bad ones, when the careless nurse will absolutely fail or the weak mother utterly despair. When the infant passes into the stage of intelligence or conscious imitation, personality counts infinitely more. The child is made up of two selves, the self of habit and the self of accommodation.† The self of habit and hereditary tendencies is the self which dominates the weaker vessel with which he comes in contact. It is the self which he "ejects" into the younger children, the weakling nurse, and the grandmother. He shows on this side the goodness or badness of his breed and education. The accommodating self is the self that learns, that imitates, that is influenced by the stronger characters around him, and which he absorbs and transfers to his habitual self. It is the business of training and education to influence the accommo-

* Baldwin, *loc. cit.*, p. 116.

† See Baldwin, 'Social and Ethical Interpretation in Mental Development,' p. 34.

dating self. The child finds himself stimulated to control his impulses and his desires by conforming to the will of a stronger and greater personality.

How much of the ultimate result, whether the training and environment be good or bad, depends upon hereditary influences we all must admit. But there are few children indeed of normal intelligence that will not be gradually brought under the influence of those with whom they come in contact. Much depends upon heredity and much on parents. How many are equal to the task? I will not say hard things of the class of parent to whom the nursery is an unexplored country and who give to billiards and bridge what was meant for mankind; rather let me insist on the harm which is done by devoted parents in the direction of over-care and over-indulgence. Every intelligent parent recognises the evils of carelessness; perhaps few think of the evils wrought to the infant or child that is never allowed to lead a natural life and is protected from every wind that blows. Over-anxiety and over-care make a bad school for neurotic children for learning self-restraint and self-control. The child quickly accepts the situation that he is the centre round which all the household moves. He soon learns that he must not be allowed to cry, and cry he does till he gets what he wants. With the laudable desire to give pleasure to the children and make them happy they are over-indulged in every luxury and every excitement that appeal to the emotional side of their nature. How great the harm done to the present-day neurotic child by a life of excitement no mortal man can assess. A world-wide authority in an address given the other day said: "Young people should go tired to bed and have no time for unhealthy excitement." And may I with no uncertain voice plead as a cure for the neuroses of the twentieth-century child hard work and hearty play, a simple and a healthy life.

(*Delivered May the 19th*, 1905.)

89. CLINICAL CASES AND PATHOLOGICAL SPECIMENS SHOWN AT THE CHILDREN'S HOSPITAL, SHEFFIELD.*

Clinical Cases.

Dr. C. N. Gwynne (Sheffield):

(1) A case of congenital dislocation of the hip, treated by the open method.

(2) Two cases of congenital dilatation of the colon.

(3) A case of multiple tubercular foci.

(4) A case of infantile paralysis of the leg, treated by arthrodesis. A case of calcaneo-valgus treated by the same method was also shown.

(5) Excision of the knee for tubercular disease.

(6) Case of (?) Hodgkin's disease.

Dr. C. H. Willey (Sheffield):

A case of pseudo-hypertrophic paralysis.

Dr. J. H. Wilks (Sheffield):

(1) Five cases of congenital dislocation of the hip.

(2) Strangulated hernia in a baby 2 weeks old. Operation. Recovery.

(3) A case of paralysis of the 3rd, 6th, and 7th nerves.

(4) A case of huge nævus. Operation refused. Recovery.

(5) A case of congenital pulmonary stenosis.

Mr. Simeon Snell (Sheffield):

(1) Cases of blindness from congenital defects.

Dr. Thomas H. Morton (introduced) (Sheffield):

(1) A case of malformation of the spine and side.

(2) A case of malformation of the hand.

(3) A case of Dupytren's contraction of the hand.

(4) A case of enlargement of the liver.

Dr. Harold Leader (introduced) (Sheffield):

(1) Three cases of Erb's paralysis.

(2) A case of splenic enlargement.

* Prior to the formal opening of the proceedings of the Provincial Meeting at Sheffield, from 2.30 to 4.30 p.m., on Saturday, June the 17th, 1905.

Pathological Specimens.

Professor Arthur Hall (introduced) (Sheffield) exhibited a series of pathological specimens from the Museum of the Medical Department of the Sheffield University, including (1) hydrocephalus, (2) tubercular tumours of the brain and cerebellum, (3) abscesses of the brain, (4) meningitis, (5) granular kidneys in childhood, (6) Hodgkin's disease, (7) meningocele, (8) cirrhosis of the liver.

Dr. J. G. Emanuel (Birmingham): Congenital obliteration of the bile ducts.

Dr. J. H. Wilks (Sheffield): Imperforate rectum.

Dr. Harold Leader (introduced) (Sheffield): Kidneys and mediastinal growth from a case of lympho-sarcoma.

90. A CASE OF OSTEOMYELITIS WITH SUPPURATIVE PERICARDITIS TREATED BY INCISION AND DRAINAGE; RECOVERY.

By C. N. GWYNNE, M.Ch.

Gertrude P—, aged 7 years, was admitted into the Children's Hospital, Sheffield, on August the 7th, 1903, suffering from osteomyelitis with pericarditis.

History.—She was quite healthy until July the 30th, when she complained of pain in the right hip-joint when she walked. Her mother noticed a swelling below the joint, and on August the 4th a smaller swelling was noticed about the left wrist-joint.

Present condition.—Pulse 120 to the minute. Temperature 102° F. Respirations 24 to the minute. The face is flushed and anxious, and the child looks very ill. The pupils are rather dilated, equal, and active. The tongue is coated. The right thigh is swollen, tense, red and tender

in front and to the outer side. The hips and knee are not involved. The right lower radial epiphysis is enlarged, red, and tender, but the wrist-joint is not involved. She answers questions, but wanders in her answers, and she is very restless.

August the 8th, 1903.—An incision was made over the lower part of the femur, and pus was found under the periosteum. An incision was also made over the lower end of the left radius, and the bone was drilled, but there was no pus in the medulla. Pus was found under the periosteum.

August the 11th.—The temperature went up this morning, and she was found to have right parotitis and pericarditis.

August the 13th.—The parotid swelling was opened. There was no discharge.

August the 16th.—Two drachms of thick cheesy pus came from the parotid wound.

August the 21st.—Dulness was detected on the left side of the chest. An exploring needle was inserted between the sixth and seventh ribs in the mid-axillary line, and pus was found. Under $CHCl_3$ a portion of the sixth rib was excised, but no pus was found. The wound was stitched up, and an incision made over the pericardium. This was opened and about ʒx of pus escaped. A rubber tube was inserted, and the wound dressed with gauze. The child became much collapsed during the operation, and an injection of strychnine was given. She recovered quickly after being put to bed.

August the 22nd.—The wounds were dressed; discharge of pus was free.

August the 26th.—The rib wound in the axilla had healed. Pus still coming freely from the pericardial wound.

August the 30th.—Tube removed from the pericardium. Heart sounds clear.

September the 8th.—Less discharge from the pericardium, more from the right leg; arm wound looking gangrenous.

September the 9th.—Poultices applied to the arm every three hours.

September the 11th.—Arm wound improving. The right leg and foot much swollen and painful. Under $CHCl_3$ an incision was made in the upper thigh; no pus found, tube inserted. An incision was then made on the outside of the leg; no pus found, and the wound was stitched up.

September the 15th.—The wound in the right leg was closed. An abscess was found in the calf of the right leg and opened.

September the 25th.—The wound last made was now healed.

October the 3rd.—Child putting on flesh and looking well lately.

November the 3rd.—The temperature to-day ran up to 103° F. Pain was complained of at the lower end of the right femur, which was relieved by hot fomentations.

December the 11th.—The cicatrix at the lower end of the right femur found to be breaking down; a probe goes in $\frac{1}{4}$th inch, but does not reach bare bone. Dressed with red lotion.

December the 21st.—Cicatrix healed.

June the 7th, 1904.—*Patient discharged.* All wounds healed. Heart sounds perfect.

April the 5th, 1905.—Patient re-admitted into hospital. Since leaving the institution she has been perfectly well and going to school regularly. Ten days ago she complained of pain over the lower end of the right femur and in the popliteal space. Three days after that the old wound at the lower end of the femur broke down, and has discharged ever since. A second opening appeared in the popliteal space, and this also discharged.

On admission.—Patient appears to be in excellent health. She complains of pain above the right knee, which is red, swollen, and tender. There are three discharging wounds—one on the outer side, one on the inner side of the lower end of the femur, and one in the popliteal space. There are old cicatrices at various parts of the body—one over the lower

end of the left radius, one over the 6th rib in the axillary line, one over the pericardium, one over the upper and outer side of the right thigh, one over the upper and outer surface of the right leg, and one behind the calf of the right leg. All these are quite sound. The heart's apex beat is visible and palpable in the sixth interspace about ½ inch inside the nipple line. It lies just beneath the old cicatrix. The heart sounds are pure and good; there is slight irregularity in rhythm. The lungs are normal. Hot fomentations were applied to the lower end of the right femur.

April the 10th.—The fomentations have removed all pain and swelling from the leg. Under $CHCl_3$ the discharging wounds were examined and the openings enlarged. Bare bone was easily reached in all directions, but as the sequestrum was not loose it was left alone. The sinuses were scraped and plugged.

April the 19th.—All the wounds are quite healed now.

(*Read at the Provincial Meeting, Sheffield, Saturday, June the 17th,* 1905.)

Discussion.

Mr. H. Betham Robinson said the point which occurred to him as of interest in the case narrated by Dr. Gwynne was the associated complication of parotitis. He believed it to be the general opinion that the bulk of the cases of parotitis were seen in relation to abdominal disease, but at the present time it was open to doubt if there was any special relation to the abdominal lesions, but rather that the parotitis was dependent upon some septic condition inside the mouth. He asked whether there was anything in the present case pointing to such; was the mouth in a nasty condition? If not, was there any other particular reason for the parotitis?

Dr. C. N. Gwynne (Sheffield), in reply, said that during the child's very septic condition the tongue and the mouth generally were in a very nasty state, but he thought there was nothing present to justify the belief that the parotitis was due to that.

91. A CASE OF TUBERCLE OF THE CHOROID, PRESENTING CERTAIN CLINICAL AND PATHOLOGICAL PECULIARITIES.

By GEORGE CARPENTER, M.D.,

AND

SYDNEY STEPHENSON, C.M.

In our collective experience tubercle of the choroid is scarcely sufficiently uncommon to justify the publication of a single case. At the same time, the one we are about to relate possesses some clinical and pathological peculiarities marked enough to warrant a brief account of its main features.

The facts are as follows:

Ada S—, aged 3 years, was admitted into the North-Eastern Hospital for Children, London, on July the 23rd, 1903, suffering from "bronchitis and convulsions." According to the history, the child had been well until about two weeks before admission, when she was noticed to breathe noisily at night and to be short of breath during the daytime. The personal history was unimportant, except, possibly, for the fact that the child had contracted measles when 12 months of age. The parents were healthy. There was no family history of tubercle, although it is somewhat significant that the other children in the family are said to be subject to bronchitis.

Upon admission.—The child, whose weight is 24¼ lbs., has a slight cough, but, beyond a few rhonchi at the base of each lung, no physical signs can be made out in the chest. Temperature 101° F., pulse 124 to the minute, respiration 52 to the minute.

Progress and termination.—July the 29th, 1903: The spleen can be felt to extend 1½ fingers'-breadths below the costal margin. The child now weighs 24 lbs., or a decrease of 4 ozs. as compared with the weight on admission into hospital. August the 4th, 1903: The child's temperature

since admission has ranged from 97·6° F. to 103° F., and has usually shown a tendency to rise at night to within 102° F. and 103° F. Respirations have varied from a minimum of 32 to a maximum of 76 per minute. The pulse-rate has been from 100 to 160 per minute, and it may be noted that the rate, as a rule, has been somewhat less at night than in the morning. The respirations are rapid and somewhat laboured, and the slightest exertion brings on dyspnœa and cyanosis. Resonant note all over the chest; a few rhonchi and râles can be heard. August the 6th, 1903: Beyond a few bronchitic accompaniments little to be heard in chest, which is resonant. The respirations are very frequent (64 to 92 per minute) and out of all proportion to the physical signs that are present. The liver and the spleen are slightly enlarged. Temperature 100° F. to 102° F.; pulse-rate 152 to 168 per minute. Ophthalmoscopic examination revealed the following conditions: Neither optic papilla is well defined; retinal veins large and somewhat tortuous; retinal arteries relatively small and inconspicuous. Each fundus oculi (see Plate III) contains numerous fawn-coloured tubercles, ranging in size from those that are almost equal to the optic disc in diameter to others having a diameter about equal to one quarter of the disc. They are round or oval in shape, and are discretely arranged almost exclusively in the neighbourhood of the optic disc and yellow spot—that is to say, in the so-called "central region" of the fundus. The yellow spot region itself is not invaded. Several of the tubercles are crossed by the retinal vessels, which manifest no local changes. The number of individual tubercles in each eye was computed as twelve. Close examination by the direct method showed that the fawn-coloured surface of individual tubercles was stippled with fine retinal pigment which, for that matter, was present over the unaffected parts of the fundus.

In face of the foregoing discovery, a diagnosis of acute miliary tuberculosis was made upon this date (August the 6th, 1903).

The child died comatose on August the 11th, 1903—that is to say, thirty-three days after the beginning of the illness, nineteen days after admission into the hospital, and five days after the diagnosis was made.

Autopsy.—The post-mortem examination, made twenty hours after death, revealed typical acute miliary tuberculosis, affecting specially the lungs, spleen, and choroid. The protocol was as follows: The lungs were very œdematous and studded with numerous miliary tubercles, but neither yellow nodules nor caseation were seen. The pleura was free from tubercle. A few tubercles were found in the pericardium. Although the bronchial glands were not much enlarged, yet some of them included tubercles. The bronchi contained mucus in some quantity. The larynx and trachea were free from morbid changes. The brain was normal, except for the presence of a few fine miliary tubercles in the Sylvian fissure and on the cerebellum. Miliary tubercles were also found in the spleen, kidneys, and liver, the latter of which showed fatty changes. The mesenteric glands were not enlarged. There were several ulcers (? tuberculous) on the external surface of the ileum, although none were found in its mucous membrane. Bladder, uterus, ovaries, and peritoneum were free from tubercle.

Pieces of the lungs and spleen were removed for microscopic examination. The eyes were placed unopened in formol, 10 per cent.

Microscopical examination.—(*a*) The *spleen* presented a typical picture of tuberculosis, being literally riddled with many giant-cell systems of the most characteristic kind. Tubercle bacilli were found. (*b*) The *lungs*, or at least those parts submitted to the microscope, also revealed numerous miliary tubercles. The intervening areas were consolidated. The consolidation consisted of cellular products in the alveoli. Some of the cells were of very large size, but for the most part were of moderate diameter with single nuclei. The alveoli in many cases contained also cells with horse-shoe shaped and multiple nuclei. In parts

the alveoli were stuffed with fibrin plugs which contained but few cellular elements. Infrequently a combination of the catarrhal and the fibrinous process was noticed. The alveolar walls were thick, proliferating, and engorged. The bronchial tubes had shed their mucous membrane and the smaller ramifications were stuffed with cellular products. Tubercle bacilli were demonstrated without difficulty. (*c*) The *eyes* were frozen, and bisected in a plane parallel to the horizontal meridian. Each hemisphere thus obtained was divided into an anterior, a middle, and a posterior third, each of which was then imbedded in celloidin and cut for the microscope. Slight inflammation of the optic nerve was present, as evidenced by the swelling of the papilla, the increased number of nuclei and of papillary vessels in the nerve, and the exudation of inflammatory products into the intersheath space.

Beyond this neuritis, the only ocular lesions found were those characteristic of acute disseminated miliary tuberculosis, namely, local accumulations of small round cells, with a few giant cells of the Langenhans type, situated in the thickness of the choroid, and free from pigment. Some of the choroidal tubercles showed more or less central areas of caseation, as evidenced by the absence of defined elements and imperfect or peculiar reaction to such stains as hæmatoxylin, eosin, acid fuchsin, and picric acid. Some of the choroidal tubercles showed vertical or transverse sections of the choroidal vessels, the walls of which had undergone endarteritis.

There were two pathological curiosities about the specimens. In the first place, there appeared to be in places a general infiltration of the choroid with small round inflammatory cells; and, secondly, a few outlying areas of tubercle of the retina were found.

Plate IV (Fig. 1) represents a typical tubercle formation in the choroid. A fusiform mass of small round cells occupies practically the entire parenchyma of the choroid between the lamina vitrea, which is intact, and the suprachoroid, which is also preserved. Towards the

PLATE III.

Illustrating Dr. George Carpenter and Mr. Sydney Stephenson's Case of Tubercle of the Choroid, presenting certain clinical and pathological peculiarities.

Fig. 1.—Right eye, erect image, Ada S—, aged 3 years. Disseminated tubercle of the choroid, confirmed pathologically. The child died from general miliary tuberculosis, August the 11th, 1903.

Fig. 2.—Left eye, erect image, Ada S—, aged 3 years. Disseminated tubercle of the choroid, confirmed pathologically. The child died from general miliary tuberculosis, August the 11th, 1903.

Fig. 1.

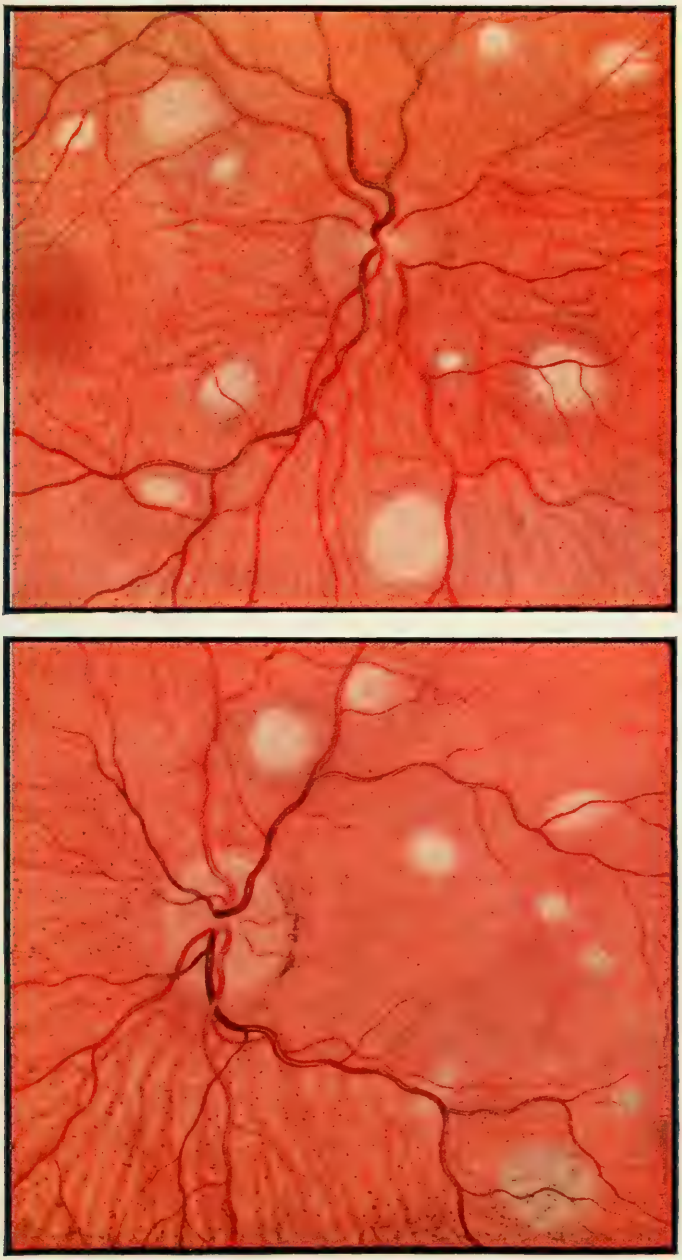


Fig. 2.

Dr. George Carpenter's and Mr. Sydney Stephenson's Case of Tubercle of the Choroid.

Adlard & Son, Impr.

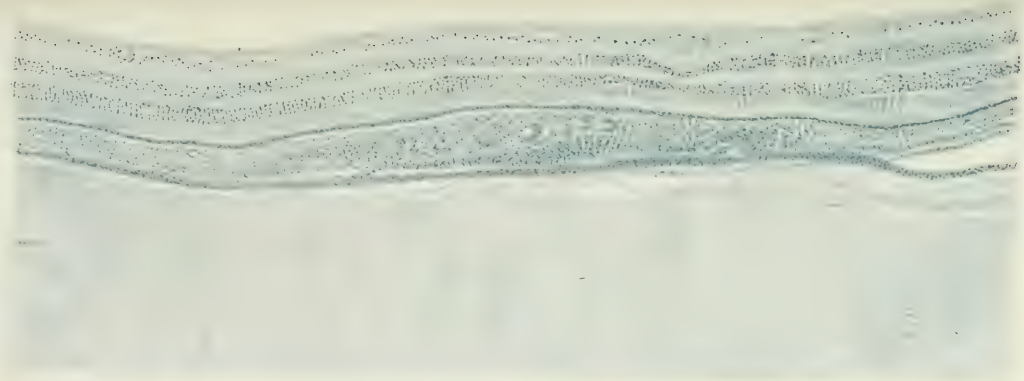


Fig. 1.—Microscopic section of a typical choroidal tubercle and infiltration of the choroid with small round cells—diffuse tuberculous choroiditis. × 50.

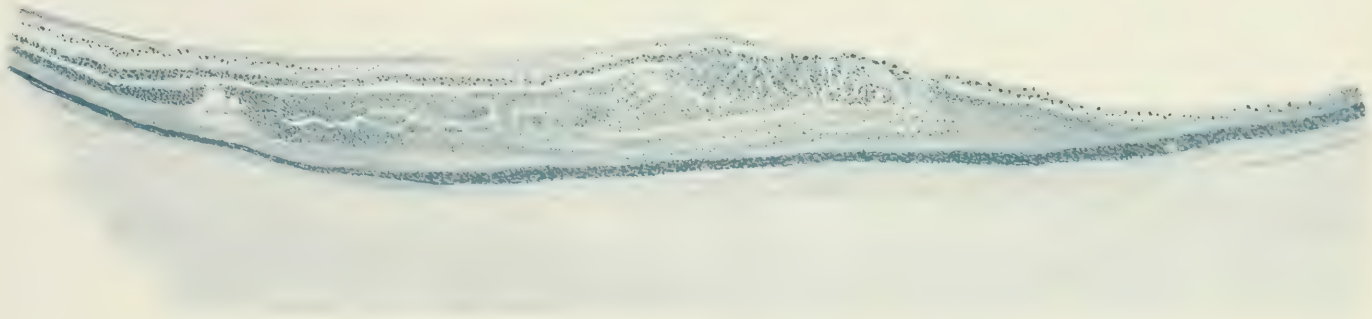


Fig. 2.—Microscopic section of a retinal tubercle. × 50.

Dr. George Carpenter's and Mr. Sydney Stephenson's Case of Tubercle of the Choroid.

Adlard & Son, Imp.

centre of the tubercle lie six giant cells, the nuclei of which are arranged towards the periphery. It will be noticed that the pigment layer of the retina is in place. A close examination of the choroid beyond the tuberculous formation will convince the attentive observer that the choroid contains a number of small round cells. In other words, in this particular case a kind of general tuberculous choroiditis is present, in addition to the simple deposition of miliary granulomata. A few of the sections showed outstanding nodules of retinal tuberculosis—*i. e.* nodules occurring independently of the choroidal deposits. Plate IV (Fig. 2) represents one of these nodules. It is sharply bounded externally by the retinal pigmented epithelium, here cut rather on the slant. The inner nuclear layer can be traced over the nodule almost in an unbroken layer. The nodule itself is composed of elements almost identical with the neurons of the outer nuclear layer, with which, indeed, it appears to be structurally continuous. No other elements can be recognised in the nodule, a point that at once raises the question whether it is to be regarded as tuberculous at all. Whatever hesitation we might have felt upon this point at first was conclusively laid at rest later by finding a couple of tubercle bacilli in one of the retinal nodules. Tubercle bacilli were also demonstrated in several of the choroidal deposits in more or less close relationship with the giant cells. The anterior part of the eye, including the iris and ciliary body, included no tuberculous deposits.

Comments.—This case illustrates once again a fact that can scarcely be too widely recognised, namely, that by far the most trustworthy sign of acute miliary tuberculosis is furnished by the discovery of tubercles in the choroid coat of the eye. As regards the present case, the progressive wasting, the oscillating temperature, the rapid pulse, the cyanosis, and especially the hurried respirations without adequate physical causes, undoubtedly pointed to acute tuberculosis as the probable underlying disease. The condition, however, could never have been diagnosed with certainty on that slender evidence. The discovery of

tubercles in the choroid settled the matter once and for all, in a way that nothing else could possibly have done. Despite recent statements to the contrary, we repeat that tubercle of the choroid is not uncommon. We recall the fact that in a former communication upon the subject ('Reports,' vol. i, 1901, p. 169) we found it to be present in exactly 50 per cent. of the forty-two cases of acute miliary tuberculosis and tuberculous meningitis that we examined with the ophthalmoscope. It is, therefore, not only a certain but also a common sign of the conditions named.

The present case, as we have already said, presents two peculiarities, viz. a clinical and a pathological one. The usual clinical picture of acute tubercle of the choroid, as we have pointed out before (*loc. cit.*), is that of one to three round, oval, or reniform areas, of usually fawn colour, and not sensibly raised, situated near the optic disc and macula lutea. The characteristic lesion, as we then stated, is generally small, single, and limited to one eye. The case just related is therefore altogether exceptional, since the choroidal deposits are present in both eyes, are multiple, and, speaking broadly, are of unusually large size.

The pathological invasion of the retina by tubercle is, in our experience, most uncommon. We have searched through dozens of microscopical specimens, taken from upwards of twenty instances of tubercle of the choroid, without having succeeded in finding this change, except in specimens taken from the present patient. It may be safely assumed, therefore, that foci in the retina in association with miliary tuberculosis of the choroid are anything but common. An even more uncommon finding would be a deposit of tubercle in the retina apart from any choroidal change. Yet such cases have been reported, amongst others, by W. A. Brailey (2), O'Sullivan and Story (3), Sattler (4), and, quite recently, by A. Knapp (5), Spalding (6), and Hancock (7).

References.

(1) Jessop, 'Trans. Ophthalmological Society,' vol. xxiii (1903), p. 50.

(2) Brailey, W. A.

(3) O'Sullivan and Story, 'Trans. Royal Academy of Medicine of Ireland,' vol. xvii, p. 451.

(4) Sattler, 'Von. Graefe's Archiv.,' xxiv, 3, p. 227.

(5) Knapp, A., 'Archives of Ophthalmology,' vol. xxxii (1903), p. 22.

(6) Spalding, 'Trans. American Ophthalmological Society,' vol. x (1903), Part I.

(7) Hancock, 'Royal London Ophthalmic Hospital Reports,' vol. xvi, Part II, March, 1905.

(*Read at the Provincial Meeting, Sheffield, June the 17th*, 1905.)

92. DEATH AFTER INSERTING A NEEDLE FOR SUPPOSED EMPYEMA.

By JOSEPH H. WILKS, B.C.

The point of interest in the following case consists in the fact that the insertion of an exploratory needle into the left side of the chest was almost instantly fatal.

The facts are as follows:

The patient, a female child, aged 3 years, was admitted into the Sheffield Children's Hospital on April the 14th, 1905, with a history that six months previously she had had measles, from which she had never properly recovered; but with the exception of this illness she had always been a healthy child. On admission her temperature was 101° F. She was sufficiently plump and well nourished, and did not seem distressed in breathing, but was troubled with a peculiar "brassy" cough, and there was slight clubbing of the fingers.

Examination of the chest showed a fair amount of movement of the thoracic wall, but it was less on the left than

on the right side. Pulsation was unduly marked around the apex of the heart over an area of about 1½ inches, and there was a faint mitral systolic murmur. The apex beat was in the fifth space, just outside the nipple line. There was dulness to percussion all over the left side, being especially evident in the supra-scapular region.

On auscultation the respiratory sounds on the left side were nowhere well heard, were universally weak, and had a soft "blowing" character, with prolonged expiration. Over the lower two thirds anteriorly they were scarcely heard at all.

Posteriorly they were "tubular" all over and very weak, and no crepitations were detected. On the right side the percussion note appeared to be normal, but the breath sounds were unduly harsh. The chief feature of the respiratory sounds was their weakness and soft blowing or tubular character.

The next day, the physical signs being the same, an exploring needle was inserted by the house-physician by my request and in my presence. No anæsthetic was used, and the child was kept lying down. The puncture was made in the eighth space, just below the angle of the left scapula, and a few small flakes of "curdy" pus were withdrawn, with some blood. Immediately afterwards blood, of a darkish hue, was seen to be issuing in a continuous stream from the needle, and almost simultaneously the child gave a cough, and expelled some blood from its mouth on to the pillow. The needle was instantly withdrawn and a finger placed over the site of puncture. The pupils dilated, the sphincter ani relaxed, the lips changed colour, and no efforts at respiration were made. The child appeared to be moribund at once, and it was apparent from the first that its condition was extremely grave. The fauces were cleared, and artificial respiration commenced, and a few gasps were produced, but no proper inspiratory effort. Five minims of adrenalin solution and gr. $\frac{1}{60}$ of strychnine were injected hypodermically. The foot of the bed was elevated, and cloths wrung out of hot water applied to the

precordium. The adrenalin injections were twice repeated, and saline solution given *per rectum*. The artificial respiration was continued for almost an hour, but from the first no sign of recovery was shown. It was thought that an intercostal vessel might have been wounded; as against this, percussion of the chest showed no alteration of the dulness; but on the other hand, through the abdominal wall an increasing fulness, amounting to a slight bulging in the epigastric region, was plainly visible, suggesting the entrance of blood into the stomach either through a puncture or through being swallowed. For a short time blood continued to flow passively out of the mouth, and then from the nostrils in addition, becoming of a darker coffee-ground colour.

At the post-mortem examination two days later, the larynx, trachea, heart, and lungs, were removed *en masse*. The left pleura was found to be adherent to the chest-wall all over, and also to the diaphragm. There were a few small clots on the surface of the lung at the site of puncture. The left lung appeared appreciably smaller than the right, and to have a firmer consistency; so that it roughly retained the shape and moulding of the pleural cavity. The area of the heart left uncovered by the retraction of the lung was especially noticeable, and the lung itself had numerous small grey tubercles dotted all over it. The site of puncture by the needle was seen about an inch from the base of the lung whose substance it had penetrated for a short distance. Pressure at this point caused blood to flow out, and section through the puncture showed an absence of healthy lung-tissue which had been partially replaced by more solid and indurated areas containing a number of small caseous foci. The smaller bronchi were occupied by blood-clots and puriform exudations, which were easily dislodged by squeezing. There were no blood-clots in the upper lobe, but the larger bronchi and trachea were full of blood-stained frothy mucus.

Professor Arthur Hall, of University College, Sheffield, made a microscopical examination of the heart and left

lung, and reported as follows : "Section of lung is tuberculous, with well-marked peri-bronchial inflammation, and a more or less irregular broncho-pneumonia. The heart appears to be in a condition of cloudy swelling of the muscle-fibres, but there is no evident fatty degeneration."

The lower lobe of the right lung was engorged and appeared to be in an early stage of red hepatisation, and there were also a few scattered miliary tubercles. Situated at the bifurcation of the trachea was a much enlarged gland, occupied by softened disintegrating caseous material. *The stomach* contained a quantity of dark altered blood, part of which was in clots. It was estimated that the quantity of blood lost did not amount to more than six ounces.

Pericardium.—This was almost completely left uncovered by retraction of the lung. It contained about 1 oz. of clear serous fluid. Heart-muscle pale and yellowish in colour, and flabby, weight $2\frac{1}{4}$ oz. Right ventricle dilated. A few small whitish nodules were noticeable along the free edge of the mitral valve, one of these being distinctly larger than the others, and being situated on the middle cusp.

Liver.—A few small grey tubercles on the surface of the liver, normal on section.

Spleen.—Post-mortem lividity on the posterior surface at the lower extremity, a milk spot the size of a sixpence on its anterior surface. Numerous small grey tubercles all over it.

Kidneys.—Right pale, capsule stripped off easily, a blood-clot over the right supra-renal capsule and upper extremity of the right kidney (probably due to the artificial respiration used at the time of collapse). Left kidney in a similar condition.

Descending colon and sigmoid flexure.—Collapsed and contracted.

Intestines.—Not examined in detail.

Mesenteric glands.—Enlarged.

Brain.—Not examined.

Six or seven similar cases have been recorded in the 'Lancet,' in 'St. Thomas's Hospital Reports,' and in the

'American Journal of the Medical Sciences,' and a brief but good summary is given in an editorial "On Sudden and Unexpected Death in Children," contained in the January number for this year of 'The British Journal of Children's Diseases.'* In these cases death is variously given as being caused by reflex inhibition of cardiac and respiratory centres, due to irritation of some small branches of the vagus by the exploring needle, or as being caused by asphyxia. In the case just related death, in my opinion, resulted, not from asphyxia, but from cardiac failure in a patient suffering from and in a debilitated condition due to tuberculosis with chronic or fibro-caseous changes in the lungs.

For much of the above I am indebted to the careful notes of Dr. Margaret Duncan, House-Physician to the Children's Hospital, Sheffield.

(*Read at the Provincial Meeting, Sheffield, June the* 17*th*, 1905.)

Discussion.

The Chairman (**Dr. George Willey**) (Sheffield) remarked that such cases were of the very greatest importance, whether from the surgeon's or from the physician's point of view. He hoped the Society would have the opinions of various members on the subject.

Dr. G. A. Sutherland said it was very interesting to hear the history of such a case as had just been related, both to those who had and those who had not had a similar accident. He had not had experience of a fatal result from puncture of the chest. None of the various theories enunciated in explanation of the accident seemed exactly to meet the difficulties of the case. The idea of the puncture having caused some irritation of the vagus commended itself to him (Dr. Sutherland). It was well known that the vagus could act in a powerful way upon the cardio-respiratory centre, and it was possible the needle might have irritated some sensitive terminal nerve in the lung and thus caused the reflex shock which lead to death. The mode of death in Dr. Wilks' case was evidently not one due to hæmorrhage. He asked whether the condition of the thymus gland and the glands generally was gone into at the post-mortem examination, because there was a condition of lymphatism which

* See also Editorial, "Exploratory Puncture of the Chest—Abuse of the Needle—A Warning," 'The British Journal of Children's Diseases,' October, 1905.

was also associated with sudden death from the slightest cause, and one could understand it might be a cause of death in a case of puncture of the pleura. There was a case in Vienna in which a hypodermic injection was made and the patient died suddenly, and that was explained by the Germans on the ground that the child was suffering from lymphatism. A number of cases of sudden death following puncture had been recorded without any post-mortem examination, but his belief was that they were of absolutely no value clinically. He would refer to a case of abdominal puncture in a patient who had a very large swelling of the liver. As there were some cyst-like swellings present, one of them was punctured. Nothing but blood-stained fluid came away. Two hours later he was surprised to receive a telephone message saying the patient was dead. A post-mortem examination was made, which he saw, and the abdominal cavity was found to be full of blood. The condition was sarcoma of the liver, and a softened area had been punctured, and the patient had bled to death into the abdominal cavity. Unless a post-mortem examination had been made there would have been uncertainty about the cause of death.

Mr. R. Clement Lucas said that in order to get at the truth there should be opposition to any proposal or suggestion advanced as an explanation, and therefore he took the other view, namely that in the present case there was evidence that the quantity of blood and mucus found in the lungs and stomach was sufficient to account for death. He thought that probably the needle struck some vessel of considerable size communicating with a bronchial tube, that the blood passed into the bronchial tube on the left side and came up into the throat, and so escaped to some extent from the mouth, and passed down into the other tube and into the stomach. He believed the author's suggestion that death was due to cardiac failure was not sufficient, because if it had been due to cardiac failure there would have been no bleeding.

Dr. George Carpenter said that as death from an exploratory puncture had occurred in a child under his care, which he published in the 'American Journal of the Medical Sciences,' and which he believed to be the first case on record of such an unfortunate accident, he would narrate the circumstances. Some years ago he saw a child in the Out-Patient Department of the Evelina Hospital suffering from cirrhosis of the lung, and after a careful physical examination he concluded there was no fluid in the pleura. He sent the case into the wards and expressed the opinion that there was no fluid. But his advice was not followed, and the chest was explored by the resident medical officer. Immediately it was carried out the child coughed, blood began to pour from her mouth and nostrils, and she quickly died. Post mortem there was found a cirrhosed lung.

The stomach was full of blood, the bronchi were filled with blood-stained mucus, which extended on the right side as far as the medium-sized bronchi, and on the affected side into the smallest tubes. His impression was that that child died from asphyxia, having been suffocated by the blood, and not from any effect of the puncture of the vagus, or from syncope from loss of blood. Ever since then his ideas about the security of exploratory puncture of the chest had changed very much. He did not now look upon punctures of the chest as something to be lightly undertaken. He did not wish to convey the impression to the Society that he did not use this valuable means of diagnosis frequently. On the contrary, he used it very frequently indeed, but before exploring he was always most careful to make a thorough examination. He was under the impression that younger members of the profession when they were not quite certain what was inside the chest were in the habit of puncturing first, and conducting a careful physical examination afterwards. He (Dr. Carpenter) not very long ago related the just narrated unfortunate experience to his house-physician, who was an advocate for exploring chests on the slightest provocation, and suggested to him (in view of this) that it would be better to postpone the exploring of chests under his (Dr. Carpenter's) care to the times of his periodical visits. He could see the house-physician was not impressed by this, and he no doubt regarded him as old-fashioned and over-anxious. Next day, however, the house-physician explored the chest of a patient under the care of a colleague at the North-Eastern Hospital for Children, but on this occasion, fortunately, in the presence and by the authority of the physician. The child almost immediately turned pale, a few drops of frothy blood appeared in the mouth, and death quickly ensued. Post mortem the stomach was found to be full of blood. The lung was cirrhotic. On one or two occasions he had been startled by the appearance of small quantities of blood in the mouths of children, and by pallor. True, they had not died, but it was not a pleasant experience. Fortescue-Brickdale narrated in the early part of the Society's session the case of a rickety child whose chest was explored for a supposed empyema. Almost immediately a little blood was coughed up, cyanosis appeared, and the child died about ten minutes after the little operation. In this case, unfortunately, a post-mortem examination was not obtained. The lesson which the cases just related taught him (Dr. Carpenter) was that one must be very careful in all chest explorations, and he thought that needling a cirrhotic lung was especially dangerous, and should always be avoided.

Dr. Edmund Cautley said he fully agreed with the remark of Dr. Carpenter that exploration of the chest should not be lightly undertaken; but he strongly held that it was necessary to explore in children very frequently, because it was absolutely

impossible in many cases to diagnose on physical and clinical grounds between the presence of fluid and consolidation, or collapse. Personally, he was not afraid of exploring the chest. After many years' experience, and after supervising it in the hands of numerous resident physicians, he had never seen an accident occur. It was extremely interesting to hear Dr. Carpenter's experience in addition to that of Dr. Wilks'. He was very sceptical about the possibility of exploratory puncture of the chest producing sudden death from reflex action. He thought the explanation given in the present case was quite feasible, that it was due to hæmorrhage and asphyxia. In the other cases on record where death had suddenly followed exploration, and where nothing had been found to account for death, he inclined to the view which Dr. Sutherland had hinted at, that the death was probably due to lymphatism. He could not believe that simple exploratory puncture of the chest could produce such fatal shock, even if one irritated the vagus nerve, as to cause sudden syncope.

Mr. Joseph H. Wilks (Sheffield), in reply, said attention had not been directed to the enlargement or otherwise of the thymus gland, but the post-mortem record showed that that gland was not enlarged. The mesenteric and bronchial glands were enlarged, but only those. With regard to the cause of death, it occurred to him that the loss of blood certainly had something to do with it, but he regarded this only as an adjuvant; he thought the heart was inclined to fail, and the loss of blood would probably increase that tendency and prevent recovery. Moreover the mode of death was not such as one would expect from asphyxia. The patient lay quiet from the first, and there was no convulsive movement, and though there might have been a transient darkening of the lips there was no real cyanosis and no effort on the part of the chest to get rid of any blood, such as one might expect in a case of blocking of the bronchial tubes.

93. NOTES OF A CASE OF LYMPHO-SARCOMA.

By HAROLD LEADER, M.B.

(Introduced.)

A. L—, aged 11 years, admitted into the Children's Hospital, Sheffield, on December the 14th, 1904. He had been quite well up to two months before admission, being "fat and rosy." It was then noticed that he had swellings under

the jaw on both sides, and smaller "lumps" all down his neck. He complained of pain across the abdomen, and it was noticed that he was getting thin and pale. He has vomited frequently, and his bowels have been confined. Urine has been passed in large quantity, and he seemed very thirsty.

Previous history.—Has had pertussis and attacks of "bronchitis" in winter.

Family history.—Youngest of five children. Mother had two miscarriages, and one child born at the eighth month.

Condition on admission.—Child very thin and anæmic. Lymphatic glands enlarged on both sides of the neck, the largest under the jaw being about an inch in length. Smaller glands could be felt in the posterior triangles of the neck. These were moderately hard, discrete, and movable under the skin. The glands in the axillæ and groins were slightly enlarged. The abdomen was full. The liver came to three fingers'-breadth below the costal margin. The spleen could not be felt. There was a large tumour on each side of the abdomen, the rounded lower end of which came to below the level of the umbilicus; they felt like renal tumours. No abnormal physical signs could be detected in the lungs, and the heart was apparently healthy.

Urine.—Clear, pale yellow; specific gravity 1014, no albumin or blood.

December the 21st.—In a fresh specimen of blood there was a decided increase in white corpuscles.

Differential count.—Small lymphocytes, 66 per cent.; large mononuclear, 3 per cent.; polymorphonuclear, 27 per cent.; myelocytes, 4 per cent.; nucleated red corpuscles, 4 in 100 white; no poikilocytosis.

February the 9th, 1905.—Patient seemed weaker and cried easily. The glands in the neck had become larger, but not to any great extent. The face was swollen and shiny. Mouth very septic, and gums swollen and soft. Weight 3 st. 10 lb. 4 oz.

Examination of blood.—White corpuscles, 21,500 per

c.mm.; red corpuscles, 4,320,000 per c.mm.; hæmoglobin, 70 per cent.

Differential count.—Small lymphocytes, 64·5 per cent.; large mononuclear 16·1 per cent.; polymorphonuclear, 19·04 per cent.; leucocytes (Ehrlich), 0·2 per cent.; nucleated red corpuscles, 6·6 in 100 white corpuscles; megaloblasts 2 in 378 white corpuscles.

February the 17th.—Mouth cleaner; face puffy, especially under the eyes. Weight, 3 st. 9 lb. 8 oz.

February the 20th.—*Urine*, pale, acid; specific gravity, 1010; albumin, 2 per cent.

February the 23rd.—Patient very weak, gums spongy. Weight, 3 st. 11 lb. 5 oz. Some œdema of the feet and legs had now appeared.

February the 24th.—Patient complained of sharp pain in right side of the chest, and he became very pallid and dyspnœic.

February the 25th.—Very ill and blanched. Temperature 102° F. Pulse rate 120 to the minute. Respiration rate 32 to the minute. Nares working; great difficulty in breathing; pain in right side of chest; a few dry râles could be heard on the right side in front, but the back was not examined. Died at 10 p.m.

Urine passed just before death contained no albumin; some crystals of uric acid were seen under the microscope. The temperature had been subnormal until just before death. The fundus oculi showed no pathological changes.

Treatment.—Arsenic was given only for a short time, attempts having been made to get the tongue and mouth into a cleaner condition.

February the 27th, *Autopsy.—External appearances*: Body considerably emaciated, abdomen distended. Small petechiæ on the outer side of the legs and at the upper part of abdomen. There was a bruise at the lower part of the left tibia.

Abdomen.—On opening the body the liver was seen to come three fingers'-breadth below the costal margin. The stomach was dilated and distended with gas. It showed

small hæmorrhages in its wall. The intestines were dark green from decomposition.

Liver.—Pale, otherwise no naked-eye changes.

Spleen.—Friable, not enlarged, slight perisplenitis.

Kidneys.—Both were very much enlarged and pale, at first sight looking like large white kidneys. The capsule stripped easily, and the surface showed stellate venules on a pale surface. The right kidney weighed $17\frac{1}{2}$ oz., the left 16 oz. They both measured $7 \times 3\frac{1}{2}$ inches. The kidney substance was firm but too elastic; the pelves and calices were dilated. The cut surface had a smooth uniform appearance, cream-coloured, the pyramids appearing as areas of mottled vessels with irregular edges leading into the surrounding pale tissue. The pyramids were practically destroyed in their inner part.

Ureters were not dilated. *Supra-renal capsules* pale. *Bladder* full of pale urine. *Stomach and intestines* not opened. *Mesenteric glands* moderately enlarged, not caseous.

Chest.—On removing the sternum a large pale mass, at first thought to be the thymus, was seen lying in the anterior mediastinum. This extended from the root of the neck above and overlapped the pericardium below. It surrounded the great vessels and the trachea without diminishing their lumen. The posterior and lower aspect was funnel-shaped to receive the pericardium. The lungs or other adjacent structures were not invaded. This mass weighed 7 oz., and measured $5 \times 3\frac{1}{4}$ inches. It was very hard in consistence, and proved not to be the thymus, but a diffuse lympho-sarcomatosis of the mediastinum.

Lungs.—In the right pleural cavity there were four or five ounces of sero-purulent fluid. On the left side the lung was attached to the chest-wall by numerous fairly recent adhesions, but there was no fluid. Both lungs were œdematous, no consolidation.

Heart.—Weight 5 oz. Left ventricle hypertrophied.

Cranial cavity.—Not examined.

Microscopical appearances.—The *kidneys* showed an enor-

mous increase of small round cells, separating widely the tubules, which were dilated, and the epithelial lining of which showed signs of degeneration. There was a very scanty reticulum between the cells.

The *glands* showed the same large increase of cells, without a corresponding increase in the fibrous tissue.

The *liver* had masses of small cells between the lobules, chiefly surrounding the bile-ducts, but they were also scattered between the liver cells.

The growth in the mediastinum had the same masses of small cells as the other organs, but there was considerably more fibrous tissue. A few endothelial plates were seen scattered throughout the section.

(*Read at the Provincial Meeting, Sheffield, June the 17th*, 1905.)

94. CONGENITAL DISLOCATION OF THE HIP-JOINT: SOME PRACTICAL POINTS IN LORENZ'S MANIPULATIVE OPERATION AND THE AFTER-TREATMENT.

By J. JACKSON CLARKE, M.B.

(1) The scientific basis of the treatment is the same as that which led Lorenz to abandon Hoffa's open operation from the posterior aspect of the joint for his own open operation from the front of the joint, and it consists in the fact first established by Lorenz, viz. that in congenital hip-dislocation there is not, as was formerly believed, a shortening, but, on the contrary, a lengthening, of those muscles that have their origin on some part of the os-innominatum and their insertion in the great trochanter: in other words, in this deformity the pelvi-trochanteric muscles are lengthened; to divide them, as was done in Brodhurst's subcutaneous operation, and in Hoffa's open operation, is unnecessary and harmful. After a very extensive experience

of his own open operation, an experience embracing considerably over two hundred cases, Lorenz about 1895 relinquished his own open operation for his manipulative operation, which is a modification of one devised by Paci, combined with improvements and new features which justify our describing it as Lorenz's. In this manipulative operation the limb is placed in such a position that the head of the femur is placed in, on, or anterior to the acetabulum, the pelvi-trochanteric muscles being relaxed, and the limb is fixed in that position for at least six months, so that these muscles undergo a nutritive shortening. In each case it is prudent before operating to have a good skiagraph taken with the limb in an inverted position, and a careful preliminary study of the peculiar features of each case should be made.

(2) Stretching the shortened muscles. When the patient is fully anæsthetised the groups of muscles that are shortened must be thoroughly stretched. They are, first, the adductors; second, the hamstrings; and third, the rectus femoris, the sartorius, and the tensor fasciæ femoris.

In children under 4 years of age the adductors may sometimes be sufficiently stretched by powerful kneading of the muscles when made tense by forcibly abducting the limb, but usually they require division either by repeatedly beating the stretched muscles with the ulnar border of the hand at a point below the tendinous portion of the adductor longus. In older children I sometimes prefer to divide the prominent adductus subcutaneously by a tenotome; this measure, without risking sepsis, diminishes the subsequent swelling. The hamstrings are stretched by repeated swinging passive flexions of the hip-joint, the knee being kept extended, and these movements are continued till the patient's foot can be placed over the corresponding shoulder. The anterior muscles are stretched by successive forcible passive extension movements of the hip, the knee being flexed, and these movements are to be continued till the patient's heel can be readily brought to touch the buttock. The capsule and other fibrous tissues are

stretched by downward traction of the limb against counter-extension applied by a roller towel placed upon the corresponding tuber ischii, some cotton-wool intervening to protect the skin. Usually manual traction is sufficient, and I prefer to grasp the patient's thigh above the lower femoral epiphysis. In children of 8 or 9 years of age—*i. e.* about the limit of age when this operation can be safely carried out—it is sometimes necessary to perform this traction by a second roller towel placed in a clove-hitch about the patient's limb, and attached to a windlass or screw extension-apparatus below. In this way the great trochanter should be brought down to or below its normal level. Only experience can teach the amount of force that can be safely applied in any given case.

(3) The act of reduction. The knee flexed to a right angle and the limb inverted, the thigh is flexed upon the abdomen. At the moment of reduction a padded wedge is placed under the buttock between the tuber ischii, whilst strong passive abduction is exerted. In young children simple pressure of the finger-tips, and in older ones the clenched fist of the operator will serve the purpose of this wedge. These manipulations may require to be repeated several times, and in older children if reduction is not obtained at a first operation success may be obtained at a second or third. There is no mistaking the signs of successful reduction: the head of the femur passes forward, filling the previously abnormally hollow middle part of Scarpa's triangle, and usually there is a distinct sound or shock of reduction. I like to feel the femoral artery pulsating external to the head of the femur. The type of acetabulum present is ascertained by the sensation communicated to the hand of the surgeon whilst the head of the femur is purposely re-dislocated, first in a backward, then in an upward, and finally in a downward direction. If the margins of the acetabulum are thus found to be shallow, the greater is the need of securing a forward position of the head of the bone, as evidenced by the prominence of the head of the femur and its relation to the femoral artery

mentioned above. This is secured by pressing back the knee several times and finally holding the limb so that it makes an angle less than a right angle with the mid-lateral line of the trunk and the inner surface of the knee is posterior to the mesial frontal plane of the body—*i. e.* just posterior to an imaginary line drawn transversely through the middle of the perineum.

(4) Application of the first retention apparatus. In my experience a plaster-of-Paris apparatus, applied strictly according to Lorenz's method, is the best. This method can only be learnt by seeing it done. Whilst the cotton-wool, calico bandages, and plaster bandages are being applied by an assistant, the patient being supported in Lorenz's pelvic rest and a shoulder rest, the surgeon must with sedulous care maintain the limb in the correct position.

(5) Massage and exercises according to Lorenz's directions must be carried out during the six or eight months that the first retention apparatus is worn as well as afterwards. The entire course of treatment lasts from one to two years.

(6) Special cases. When the head of the bone has been purposely fixed in a forward position the security from re-dislocation is greatest, but a second retention apparatus to fix the limb in a position of diminished abduction, with neutral rotation (toes pointing forwards) is necessary for two or three months, and the resulting slightly flexed and stiff joint is restored to complete function by suitable exercises.

In conclusion, my experience leads me to believe that in practised hands Lorenz's manipulative treatment affords a means of restoring perfect functional and usually perfect anatomical conditions to patients afflicted with congenital hip-dislocation, and this without serious risk when the patient has not passed the age limit mentioned above.

(*Read at the Provincial Meeting, Sheffield, June the* 17*th*, 1905.)

Discussion.

Mr. A. H. Tubby said all present would be willing to express their gratitude to Mr. Jackson Clarke for the exceedingly clear way in which he had put forward the most important points in connection with the Lorenz method of reducing congenital dislocation of the hip. It was a subject which was so surrounded by difficulties of every description that no one could hope to lay down rules which would meet all the difficulties in treatment which arise. He had no criticism of the paper to offer, because the case had been put so clearly and reasonably that no room was left for adverse comment; he only desired to offer one or two remarks. With regard to skiagrams, a most important point, for which he was indebted to Dr. David Morgan of Liverpool, was to have three skiagrams taken—*i. e.* with the foot in the three positions of eversion, inversion, and with the foot straight in front. It was astonishing how different the outlines of the parts affected looked from different standpoints. Mr. Jackson Clarke had also spoken of subcutaneous tenotomy. He (Mr. Tubby) used to use that method for the adductors, but it appeared to him that the division was not all that one would desire. Therefore for the last four years he had tenotomised the adductors by the open method a fortnight before the attempted reduction, and by that means he found he obtained a more free and certain division.

Mr. Jackson Clarke, in reply, thanked Mr. Tubby for his remarks. He agreed with Mr. Tubby as to the importance of having skiagrams taken in different positions. But in cases where the cost of only one skiagram could be borne, the inverted position was the most important, because it showed the whole length of the femoral neck, and thus afforded information useful in prognosis. The only reason why he had refrained from dividing the adductors through an incision was that it was rather a hustling kind of operation, and what did not yield to the tenotome yielded to the stretching movements, and he did not like having an open wound when manipulating the parts, one result of which was to suck in the air with its contained germs. It was chiefly to exclude the germs that he favoured the subcutaneous method.

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95. A CASE OF MEDIAN HARE-LIP.

By R. C. DUN, C.M.

This patient, a girl, aged 1 year and 10 months, was born with a deformity of the upper lip for which nothing

had been done up to the time of her coming under my care, a few weeks ago.

Four other children of the family were well formed, and

FIG. 16.

neither parents nor relations had hare-lips or any kind of congenital deformity.

The following notes of the condition were taken: The upper lip is cleft exactly in the middle line. The cleft is narrow and extends upwards to a point a quarter of an inch above the free margin of the alveolar process. At this level

the mucous membrane covering the edges of the cleft is reflected on to the upper jaw, forming a distinct frenum. The right edge of the cleft is straight, the left slightly oblique. A deep groove is continued upwards in the middle line from the apex of the cleft through the upper portion of the lip, the nasal septum, and the tip of the nose to end on the dorsum. This extension of the groove beyond the point of the nose gives rise to a bifid appearance of the organ at its lower part. The labial part of the sulcus is lined with mucous membrane; the nasal portion has a normal skin covering.

The anterior nares are small and widely separated, owing to the abnormal width, half an inch, of the intervening portion of the nasal septum. The bridge of the nose is broad and flattened. The anterior borders of the nasal bones can be distinctly felt separated from each other by an interval of half an inch at their lowest points. The eyes are wide apart; the distance between the inner canthi measures $1\frac{1}{4}$ inches.

The incisor teeth are perfectly developed and regularly placed. There is no indication of any alveolar cleft. The hard palate is highly arched. The examination of the nose is difficult on account of the small size of the anterior nares, but so far as can be seen the intra-nasal portion of the septum is completely developed. The child shows no other deformity. I am indebted to Dr. Hubert Armstrong for the photograph of this case.

Since these notes were taken I have operated, paring and suturing the edges of the cleft in both lip and nose. The result as regards the lip is satisfactory, but the bifid appearance of the nose is gradually reappearing as the cicatrix contracts. By means of paraffin injection I hope that I may subsequently be able to improve the appearance of the nose.

Median hare-lip is an extremely rare deformity, and I am glad to have had the opportunity of reporting this case to the Society.

(*Read at the Provincial Meeting, Sheffield, June the* 17*th*, 1905.)

Discussion.

Mr. R. Clement Lucas remarked that the condition described by Mr. Dun was extremely rare, and there were very few cases of the kind on record. One case was published in Bryant's 'Surgery,' and Mr. Hitts had published another. It depended on the notch between the internal nasal processes of the fronto-nasal process remaining unobliterated.

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96. APPENDICITIS IN A HERNIAL SAC: A REPORT OF TWO CASES.

By H. S. CLOGG, M.S.

The two cases which I wish to report serve as illustrations of a somewhat infrequent type of appendicitis, which, although by no means confined to childhood, yet occurs with some frequency at this age, and hence merits recognition at this Society.

The notes of the cases are briefly as follows:

Case 1.—Male child, aged 3 years and 8 months. To the best of the parents' knowledge it had never suffered from a hernia. The illness began with abdominal pain, centred chiefly around the umbilicus. This was not severe; it was regarded as some intestinal disorder, and treated accordingly. The abdominal pain persisted, and some hours later attention was directed by the child's complaints of pain to the right groin. The right side of the scrotum was swollen as far as the abdominal ring. The temperature was 102° F., and the pulse-frequency accelerated. The general condition of the child remained the same, but the scrotal swelling continued to increase in size, and on the third day of the illness it was evident there was an abscess in the scrotum. The surrounding œdema extended up over the inguinal canal. The bowels had been freely opened. The abdominal pain had quite disappeared. I incised the abscess, extending the cut up over the inguinal canal. The testicle lay at

the bottom of the cavity, apparently normal. At the upper end of the cavity was the swollen, terminal portion of the appendix, lying in a hernial sac. Whether the swelling had any impulse I am unable to say; it was never tested. The true cause of the abscess was unsuspected before operation. I assumed the general peritoneal cavity was shut off and explored no further. The abscess was drained and healed in about three weeks. The appendix was not, as far as I could see, perforated. I suggested that the appendix should be removed and the hernia cured at a later date, but this was refused. I heard of the child some weeks after the operation, and it was then reported quite well, and, strangely enough, it was not noticed to have any hernia. Since this time, I regret, I have lost sight of the patient.

Case 2.—A boy, aged 8 years. When an infant he had suffered from a right inguinal hernia; for this he had worn a truss for some months, and was thought to have been cured. His illness commenced with some discomfort in the abdomen. On the following day he complained of pain in the scrotum. The right half of the scrotum was reported as somewhat full, as also the inguinal canal. The temperature was 100° F. In two days the temperature became normal, and his pain had gone, and he was considered quite well. He told me later, however, that during the following few weeks he frequently had some pain in the scrotum. This he never complained of at the time. He was then laid up with a second attack of pain in the lower part of the abdomen, groin, and scrotum. Again some swelling was noted of the right half of the scrotum, and some œdema, and the temperature was elevated. I saw the boy just after this attack of pain had subsided. The right testicle was felt at the bottom of the scrotum, apparently normal. Running up from this the cord was thickened by a rounded, cord-like structure, which appeared to pass up the inguinal canal. I could not detect any impulse on coughing. From the history of the case, the presence

of abdominal, in addition to scrotal, pain, the elevation of temperature, the presence of the rounded, cord-like structure passing up from the scrotum to the inguinal canal, and my first case being quite recent in my mind, I ventured to suggest the possibility of appendicitis in a hernial sac. This condition was verified by operation. The appendix was a long one, reaching to the top of the testicle. It was free from adhesions above, the cæcum was in part drawn through the internal ring, and the appendix severed from it in the usual way. The sac was separated above, ligatured, and the inguinal canal repaired. The appendix was firmly adherent to the sac below and to the cord, and was, with some little difficulty, separated. The distal portion of the appendix only showed inflammatory changes.

Judging from the literature of recorded cases, there seem to be two ages in which appendicitis in a hernial sac is more commonly seen. The one age is between the years of 50 and 70, and the other is in childhood. I estimate about 25 per cent. of recorded cases have been in children. In this connection two points may be noted—(1) the great frequency of hernia in children; (2) hernia of the appendix is almost, if not quite, as commonly observed in children as at any other age.

In order that the appendix may be thus engaged in a hernial sac it must be of some length, have an elongated mesentery, and the cæcum should be mobile, the latter condition being commonly seen in children.

When the appendix lies in a hernial sac it would seem to be even more predisposed to inflammation than when situated in the abdomen. It is very liable to be kinked, its lumen obstructed, and its vascular supply interfered with either by compression from without or by the contractions of the abdominal wall.

With regard to the age incidence, appendicitis in a hernial sac has been recorded in quite young infants. It has been reported at the ages of 6 weeks, 3 months, 5 months, and 8 months of age.

So far as the condition has been observed in children it

has always been in connection with an inguinal sac, and I think I am right in saying in every case the sac was on the right side. I have not found any record of a case occurring in a female child.

In children in the majority of cases recorded the appendix has been the only content of the sac. Less frequently the cæcum in part is also present.

The symptoms of an inflamed appendix occurring in a hernial sac are in general less severe than when occurring in the abdomen. This, I take it, depends in some measure on the age of the hernia and the alterations in the peritoneum forming the sac. Should the sac have become thickened, as hernial sacs do, when inflamed it will not react as normal peritoneum, and hence the constitutional effects will be less marked.

Acute appendicitis in a hernial sac has only rarely been diagnosed or suspected. There are several conditions with which it may be confounded. These may be divided into two classes:

(1) Any complication occurring in an inguinal hernia.

(2) Any inflammation of the testicle or its coverings.

One may emphatically state that such rare conditions as strangulation of the appendix, strangulation of a Meckel's diverticulum, or inflammation of the same, giving rise to precisely the same symptoms, cannot in any way be differentiated from appendicitis in a hernial sac. Most of the cases have been mistaken for strangulated herniæ. There are many points of resemblance, viz. the sudden onset, the abdominal gland, the sudden appearance of a swelling in the inguinal region and scrotum, irreducible or without impulse, or, should the hernia have been down before, its sudden increase in size. There are, however, some points of difference. In appendicitis some fever is present from the beginning: the temperature is often subnormal in a strangulation of the gut. The swelling in an obstructed hernia is rounded, well-defined, tense, and elastic: in appendicitis it is usually of smaller size, ill-defined, and the skin early shows some œdema and perhaps

redness; the child may complain of abdominal symptoms before anything is noted amiss in the inguinal region. In both conditions constipation is present, but is more absolute in the obstructed hernia. In fact, we have in one case a primary obstructive lesion, with inflammatory signs long delayed, and in the other case a primary inflammatory lesion as shown by fever, œdema of the coverings, and ill-definition of the swelling.

Inflammatory conditions of the testicle can generally be differentiated by recognising it is the testicle which is swollen. In appendicitis the testicle will be felt at the bottom of the scrotum, and normal: the upper limb of a testicular inflammation can be defined and the inguinal canal is free.

There is a further condition which may possibly be mistaken for it—torsion of the cord. Especially will this be so when, as is not infrequently the case, the testicle lies high in the scrotum or is imperfectly descended. Here it is the testicle itself which is swollen, the upper limit of the swelling may be reached and the inguinal canal is free (unless associated with a hernia, which is sometimes the case). Abdominal symptoms are absent, and the temperature in the early stages is not raised.

(*Read at the Provincial Meeting, Sheffield, June the* 17*th*, 1905.)

Discussion.

Mr. R. Clement Lucas said a paper appeared in the 'Guy's Hospital Reports' for 1883–4 by his late colleague, Mr. Davies-Colley, describing several cases, one of which was his (Mr. Lucas's), in which the appendix alone was strangulated within the hernial sac. Perhaps appendicitis was not so well known then as it is now, and some of the cases which were then thought to be strangulation of the appendix in the sac might possibly have been instances of appendicitis in a hernial sac, such as Mr. Clogg had now described. Perhaps Mr. Clogg would find some matter of interest in the 'Guy's Hospital Reports' to which he (Mr. Lucas) had alluded.

Dr. C. W. Gwynne (Sheffield) said the presence of the appendix in a hernial sac was not so very rare. Two such cases

had been in the Sheffield Children's Hospital. He asked whether Mr. Clogg thought the use of a truss had anything to do with the inflammation of the appendix in one of the cases. It seemed to be an additional argument against the use of a truss for a young infant.

Mr. H. S. Clogg, in reply, said it was six years since the truss was worn by the child, which was quite an infant when the instrument was applied. It was thought a cure had been effected, since the hernia had not been noticed since. He had not seen the paper to which Mr. Lucas referred, but would take an early occasion to read it. Some had recently doubted whether strangulation of the appendix could exist apart from previous inflammatory swelling, though at one time the symptoms of strangulation of the appendix were said to be similar to those produced by strangulation of the lumen of the intestines. A few French writers seemed to maintain this view still. In the first case the appendix might have been strangulated at the internal ring, but he did not think that. He would not have expected it would have healed up so rapidly if the whole appendix had been strangulated.

97. ACUTE OSTEOMYELITIS AND PERIOSTITIS OF THE SPINE.

By A. H. TUBBY, M.S.

These affections, generally believed to be rare, are of great importance, on account of the serious symptoms to which they give rise, and of much interest, because of the difficulties in diagnosis.

References to the literature of the subject show that the earlier records speak of exceedingly acute cases; but extended experience and more discriminating diagnosis have verified the existence of less acute and non-suppurative cases, which run a benign course as regards the life of the patient and the integrity and mobility of the spine. Records of cases dealing with this subject are to be gathered from French, German, and English literature. Some of the earliest cases were observed and accounts of them given by English observers, notably by Messrs. Ballance (1) and

Clement Lucas (2), and a valuable article was written by Messrs. Makins and Abbot (3), carefully summarising the cases published up to April, 1896, including eight which had come under their personal observation, all of which were exceedingly acute.

Three degrees of the affection are now recognisable:

(1) A comparatively mild form, from which the patient usually recovers, in which there are acute inflammation and thickening of the periosteum, which subside without, apparently, causing denudation of the bone, and ultimately leave the spine in a practically healthy condition.

(2) A severe form, involving the periosteum and superficial layers of bone, associated with considerable inflammatory softening, often with considerable deformity, which may or may not go on to suppuration.

(3) A violent form, in which the bodies of the vertebræ are the site of numerous purulent foci, or the neural arches and processes are bared and necrosed. This variety usually goes on to suppuration. Implication of the spinal membranes and medulla is a constant complication and it is, therefore, exceedingly dangerous to life.

One of my objects in bringing this subject before your notice is to draw your attention to the existence of the less acute varieties; and in order to emphasise this point I append the notes of cases of this description which have come under my notice.

The following case is one illustrating the first or mild variety of the disease.

Acute Periostitis of the Cervical Spine; Resolution; Complete Recovery.

Gladys W—, aged 4½ years, was brought to me by Dr. Garnham on account of pain in the neck, with loss of mobility and malposition of the head. The history was that four months previously the child had had a very severe attack of stomatitis, which was accompanied by much prostration, high fever, and great depression. When the acute

symptoms in the mouth were passing off a sudden onset of pain occurred in the back of the neck, radiating towards its anterior aspect. The posterior aspect of the neck became very œdematous and thickened. This œdema lasted from three to four days, but no fluctuation was ever detected. When the swelling passed away the neck was found to be stiff. At no time were the glands enlarged. When I saw the child, the impression I received from her appearance was that she was suffering from torticollis of the variety which is commonly described as the "rheumatic." The neck was stiff and rigid, the head was inclined to the right shoulder, with the chin rotated towards the left side. The loss of movement was absolute, and attempts to move the head in any direction gave rise to great pain, but all the œdema and swelling had entirely subsided. I inquired of Dr. Garnham if he had observed at the time of the illness any change in the pupils or their action, and he told me that no difference had been perceptible. All the cervical vertebræ were fixed and immobile, the normal curve forward was flattened, and some thickening could be felt about the transverse processes. A diagnosis of infective periostitis was made, and as there were no signs of suppuration a favourable prognosis was given. The treatment which was carried out was absolute rest in the recumbent position, with a pillow beneath the cervical vertebræ for three months, and then the patient was allowed to get up for half the day, wearing a leather collar.

A year afterwards I again saw the child with Dr. Garnham, and found her well. She had lost all pain and distortion of the neck; she could move the head without discomfort in every direction; the cervical vertebræ were not fixed, but were fully mobile, and the only sign of the previous trouble was a prominence of the fifth cervical vertebra.

This case ran a benign course from beginning to end, and was never accompanied by any definite symptoms of involvement of the nerves, beyond the pain, which was present for a considerable time. I am inclined to suspect that a good many cases which are called rheumatic torti-

collis, occurring in children after acute illnesses, are of this nature.

As an example of the more severe form, or the second variety of case which certainly involves the bone to a varying depth, I quote the following case:

Acute Periostitis and Rapid Softening of Bone, associated with Double Pneumonia and Necrosis of the Alveolar Margins of the Jaws; Recovery.

In 1901 I was asked by Dr. Henry Huxley to see with him a little girl, aged 5 years. Six weeks previously she had been taken ill with an affection of a doubtful nature, and her brother too had suffered in the same way. The symptoms were so anomalous that no certain diagnosis was arrived at. As influenza was rife at that time, the opinion was hazarded that this was the cause. However, double pneumonia set in, and then acute stomatitis, with gangrenous destruction of the gums, and acute osteitis and periostitis of the alveolar margins of the upper and lower jaws, particularly well marked in the incisor regions. I may add that sequestra formed, and these I subsequently removed. I regret that no bacteriological observation was made at the time.

Three days previously to my seeing the child with Dr. Huxley she had complained of pain in the lower part of her back and down her legs, and on looking at the back there was discovered a large prominence, involving the second, third, and fourth lumbar vertebræ. The little child resented any attempts to move her, but there were no signs of paralysis. On feeling the prominence the impression which was given was that the affected lumbar vertebræ had sunk bodily backwards. The spinous and transverse processes were felt to be much thickened. The temperature was not raised, and no evidence of abscess was found at any time.

From the history, the rapid appearance of the prominence, the rigidity of the muscles, the loss of

mobility, and the tenderness, it was not difficult to recognise that the case was one of infective periostitis, and the opinion was hazarded that if suppuration did not ensue the child's recovery would be entirely satisfactory

The treatment consisted simply in complete recumbency in the supine position, with a convex pillow beneath the lumbar spine, for part of the day, and for the remaining part the position was changed to the prone one, so as to assist in inducing the resumption of the normal lordosis. This treatment was carried out for three months, and then a spinal support fitted.

Eleven months afterwards a letter of inquiry to Dr. Huxley elicited from him the following remarks: "The back has gone on absolutely well, and beyond the fact that the lumbar curve is a little flattened it is difficult to detect anything abnormal. She is in every respect as healthy a child as one could wish to see."

The points of interest in these cases are the appearance of the spinal symptoms subsequently to an acute osteo-stomatitis, the sudden and unexpected onset of the spinal symptoms, the rapid appearance of deformity, particularly in the second case, its almost entire disappearance, and the nearly complete restoration of the affected region to its normal functions. The connection of oral inflammation with septic periostitis of the cervical spine has been dwelt upon by other observers, and Eichel (4) records a case which was preceded by a boil on the lower lip.

I cannot help thinking that some cases of spinal rigidity, occurring in weakly and ill-fed children, are ascribed to tuberculosis, especially if, as sometimes happens, meningitic symptoms complicate the affection. But if the history were carefully inquired into and the case thoroughly observed from the first, a few would be found to be of septic origin, and not tuberculous. So far, I have come across no post-mortem observation made on these milder cases which do not go on to suppuration. They are distinct varieties, of which, now that attention is drawn to them, no doubt further examples will be forthcoming;

and one of my objects in making these remarks is, if possible, to elicit further information and observation on the subject.

With regard to the third variety, or those exceedingly acute and violent and too often fatal cases, I have met with one example only, to which I shall allude presently. In the middle decades of the last century, and in the later ones, a good many cases of acute osteitis of the long-bones were either overlooked or were not accurately diagnosed, and it is quite possible that such an error has occurred in osteomyelitis of the spine. I venture to suggest that in all cases of children and young adults suffering from septic disorders and exanthemata a careful watch should always be kept on the spine, and frequent examinations made. Further, if the patient die, no post-mortem examination can be considered as complete unless the spine be examined in its whole length.

Since Messrs. Makins and Abbott wrote their excellent paper a good deal of information has accumulated, and I venture to give you a brief digest of it.

The cause of acute osteomyelitis is doubtless the deposit in the bone of pus-producing organisms of any kind. In twenty cases alluded to by J. R. Hunt (5) the staphylococcus pyogenes aureus was found in fifteen, the staphylococcus pyogenes albus in one, the streptococus in two, staphylococci and streptococci in one, and the Micrococcus tetragenus in one. Also in Weber's case (6) the staphylococcus was found. Now that attention has been drawn to pneumococcic osteitis and arthritis, cases will doubtless come to hand in which this organism will be found to be present, either in the articulations of the spine or in the bones.

The exciting causes are local septic disorders, such as boils, inflammation of the gums, osteo-stomatitis, acutely septic nasal and naso-pharyngeal affections, stinking mastoiditis, whitlow, a foul puerperium, or osteomyelitis elsewhere.

Chipault (7) alludes to pneumococcic and scarlatinal periostitis. In 1895 a case came under my notice of a girl,

aged 10 years, the subject of marked deformity of the cervical vertebræ, the head being deviated to the right, and absolutely fixed there. The whole of the cervical vertebræ were immobile, and had been so since an attack of scarlet fever a year previously. A discharging sinus was situated in front of the right sternomastoid, into which a probe could be passed in the direction of the upper cervical vertebræ, but no dead bone was felt at the bottom. There was no severe pain, except on attempting to move the head, and the child could walk about without requiring any support. At the time I regarded this as periostitis of septic origin.

The account of a local injury occurs in six of the forty-one cases referred to by Eichel, but the exact relationship of injury to acute bone inflammation has never been traced, although a wide experience of osteomyelitis occurring in the long-bones shows it to be a constantly antecedent condition. In the 'Guy's Hospital Reports' of 1889 I showed that if in rabbits a blow be given on the growing line of the lower end of the femur, and a culture of streptococcus then be injected into the jugular vein, pus is formed in the cancellous tissue there before it is found elsewhere. But how a blow on the skin influences the exact site of onset of the disease is not known, except it may be, as I surmise, that alterations in the secretory activity of the glands permit the development of micrococci in the sweat-ducts, whence they are carried to the bone.

Age and sex.—The disease is most common in children and adolescents, but adults have suffered, and in one case it came on even at the age of 46 years (3). All writers are agreed that males are more liable to it than females, and roughly, the proportion is two to one.

Parts of the spine affected.—Every portion, with the exception of the coccyx, has suffered, but if the three regions the lumbar, the dorsal and the cervical regions be considered, the disease is most common in the lumbar, less so in the cervical, and least in the dorsal. That is to say, the more mobile parts of the spine suffer more than the

less, thus corresponding with the distribution of Pott's disease. In the violently suppurative form one vertebræ constitutes the focus of the disease, rarely two or more; whereas in the periostitic form, whether denudation of the periosteum or collection of fluid does or not take place several vertebræ are affected.

Statistics (3, 8) dealing with the points as to whether the vertebræ or the neural arches or other parts are inflamed are of interest because of the meningitic and myelitic complications. Briefly it may be said that these are more likely to ensue when the arches or their processses are involved, and pus is formed. Pus arising from diseased bodies makes its way forward or laterally, and appears in the neck as retropharyngeal or cervical abscess; in the lumbar region it causes psoas, iliac, or pelvic abscesses, and in the dorsal posterior mediastinal abscesses. That the neural arches are more exposed to direct injury may serve to explain the more frequent occurrence of the disease in these situations in very acute cases.

Symptoms.—The symptoms vary, not only according to the degree of the affection, but according to the locality affected. The first two cases I have quoted give a tolerably fair reflex of the signs which are observed in the non-suppurative and periosteal types. There are general symptoms of malaise, with slight fever, local pain and tenderness, stiffness, loss of mobility of the area affected, with that osteophytic proliferation so often observed in subacute spinal inflammation, but rarely seen in the very acute form. No implication of the meninges or of the cord follows, although reflected pains are present from an extension of pathological processes around the nerve-roots. And these cases show a striking power of recovery, as I have already mentioned.

Very different is the clinical picture presented by the violent type of cases. The general symptoms are closely analogous to those of osteomyelitis in the limbs. The sudden onset, the initial rigor, the early delirium, the signs of general septic intoxication, rapidly deepening in

intensity, are all present and persist until pus is found and evacuated, or the patient dies, either from septic intoxication, metastatic deposits, or meningitic and myelitic complications.

The local symptoms are excessive tenderness of the affected portion of the spine, agonising pain on attempting to move the patient, marbling and distension of the superficial veins.

If the neural arches are the sites of inflammation, local swelling, with inflammatory œdema, extending over a large area, and later on fluctuation, are in evidence. But when the bodies are affected, on account of their depth from the surface, such localising signs are not to be found, and the diagnosis rests on the history of the affection, the nature of the general symptoms, and the exceedingly acute pain and tenderness, which the patient refers to some portion of his back. Later in the course of the disease the discovery of an abscess, or the onset of acute nerve symptoms, may clear up the diagnosis. Of the latter it may be said that they are more common when the neural arches are affected, and in rare instances they may be the first symptoms. One case of Morian's (9), on account of the paralysis of the legs and of the bladder and rectum coming on so early in the case, was thought to be Landry's paralysis, but the discovery of an abscess led to an accurate diagnosis.

In the earlier stages of nerve involvement there are pains in the limbs, hyperæsthesia, and paresis, with increase of reflexes. Later there follow paralysis of motion, sensation, micturition, defæcation, and convulsions.

The local symptoms necessarily vary with the region. In the cervical portion of the spine in seven cases quoted by Hahn there were present stiffness of the neck, interference with the movements of the cervical spine, and the development of a fluctuating tumour. Irregularity or sluggishness of the pupils is present when the disease is at or near the cervico-dorsal junction (3).

The difficulty of distinguishing between disease originating in the atlas or odontoid process of the axis and the

base of the skull is remarked upon by several authors (3, 10). But it really matters little as to the exact site of origin, so long as timely evacuation of pus takes place.

Marked widening of the neck from an inflammatory œdema (Makins and Abbott), severe headache, vertigo (4), convulsions (4), are valuable signs in the cervical region.

The diagnosis of disease of the bodies in the dorsal region is very obscure, whereas if the neural arches are inflamed, and more particularly if the spinous processes are the sites of disease, the signs of acute inflammation readily present themselves on the surface.

In the lumbar region flatulence and distension of the abdomen (3, 9) are prominent symptoms when the bodies of the vertebræ are inflamed, so that the difficulty may be to differentiate abdominal affections from the spinal ones, a very common confusion, and fruitful of error.

Suppuration has occurred in twenty-four out of sixty-two cases, and fifteen of these presented grave symptoms of purulent invasion of the spinal canal, with myelitis (5).

Diagnosis.—The vital importance of early diagnosis cannot be too fully insisted upon. By it alone is it possible to prevent the spread of inflammation to the meninges, the burrowing of pus, and the spread of secondary metastatic abscesses. While acute osteomyelitis of the limbs has been a most fruitful source of mistakes in diagnosis and treatment, still more so is this the case with spinal osteomyelitis, where the possibilities of complications are increased a hundredfold. Time does not permit me to record errors which have been made, but it is well to insist on certain cardinal points, namely, (1) history of sudden onset; (2) the rigidity of the spine coincidently with the onset; (3) the early appearance of nerve symptoms; (4) the rapid development of intense general illness, with signs of a full dose of septic poisoning; (5) complaints of a pain in the back should lead to a frequent examination; (6) careful examination by the Röntgen rays may be of service in localising an abscess in this connection with the bodies of the vertebræ, and Warren (11) records a case where, by

means of the Röntgen rays, an abscess between the bodies of the second and third cervical vertebræ was discovered.

Prognosis.—The prognosis in the periosteal cases is favourable, but the more violent types are very fatal. The mortality is said by J. R. Hunt (5) to be at least 50 per cent., but evidence shows that it should be placed considerably higher than this, and that about 75 per cent. would be more correct. But it should be noted that although the mortality is greater in the earlier records, it diminishes in the later ones, doubtless owing to a wider recognition of the disease and a more perfected diagnosis. In formulating a prognosis it may be said that the neural arch cases compel recognition sooner than the body cases, and therefore invite treatment earlier. But again it should be remembered that neural arch cases, if untreated, are more likely to be the cause of involvement of the spinal canal and its contents than are centrum cases.

It is somewhat striking that in the bad types of the disease deformity is not common, certainly not in more than 10 to 20 per cent., probably owing to the fact that there is no time for deformity to appear, as the patient is either well or dead before it can do so. But in the milder forms deformity is a constant sign, often transient, and one from which the patient entirely recovers.

Treatment.—It is not necessary to allude at any length to the best method of treating the periosteal cases. Sufficient rest in the recumbent position, followed by support, will do all that is necessary.

In the suppurative cases the pus must be found and let out, and in disease of the bodies of the dorsal vertebræ I venture to throw out the suggestion that Ménard's operation of costo-transversectomy affords undoubtedly the best means of getting at the pus.

(*Read at the Provincial Meeting, Sheffield, June the* 17*th*, 1905.)

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98. THE TREATMENT OF INTUSSUSCEPTION IN INFANTS.

By GEORGE WILKINSON, M.B.

(Introduced.)

The trend of surgical opinion at the present time seems to be more and more in favour of immediate laparotomy for intussusception, as soon as the diagnosis is made. Indeed, many surgeons of eminence have expressed a hope that non-operative methods of treatment may soon come to be regarded as discredited and inadmissible. The writer cannot claim an extensive experience of intussusception,

having treated eight cases only, a number quite insufficient on which to base any general conclusions. Of these eight cases, five were treated by hydrostatic pressure alone, and recovered. Two were treated by hydrostatic pressure, followed by operation, of whom one died shortly after, whilst the other made a good recovery from the operation, but died three weeks later from diarrhœa. One case was treated by immediate operation, and died. The fact that the injection method has yielded such good results has convinced the writer of the utility of the method, and made him disinclined to reject it in favour of primary laparotomy. Further, he doubts whether a study of recent statistics lends much support to operative as opposed to non-operative methods, at least in young children.

The following is a brief *résumé* of the writer's cases.

Case 1.—Male infant, aged 4 months, treated at the Children's Hospital, Sheffield. Duration of symptoms, forty-eight hours. A tumour felt in the right hypochondriac and lumbar regions. Reduced by hydrostatic pressure under chloroform. Copious liquid motion passed immediately. Recovery.

Case 2.—Male infant, aged 14 months, treated at the Children's Hospital, Sheffield. Onset of symptoms about eighty hours previous to admission. Blood and mucus passed by bowel. A tumour felt *per rectum*. Treated by hydrostatic pressure. Tumour reduced, bowels freely moved soon after. Recovery.

Case 3.—A male infant, aged 5 months, treated at the Royal Hospital, Sheffield. Symptoms for eight hours. A tumour in the right hypochondriac region. Treated by hydrostatic pressure, and recovered.

Case 4.—A male infant, aged 13 months. Symptoms for twenty-four hours. A tumour felt through the abdominal wall in the position of the descending colon. The end of the intussusception could be felt on rectal examination two

inches within the anus. Reduced by hydrostatic pressure, and recovered.

Case 5.—A male infant, aged 7 months. Symptoms for seventy hours. Blood passed *per anum*. Had castor oil twice. Much collapsed. No tumour felt through the abdominal wall, but felt *per rectum* with child under chloroform. Reduced by hydrostatic pressure. Made a rapid and complete recovery.

Case 6.—Male infant, aged 8 months. Symptoms for seventy hours. Blood and mucus by the bowel. Tumour not made out. Owing to the absence of palpable tumour it was decided to perform laparotomy without preliminary injection. There was some difficulty in finding the intussusception, as it was tucked away under the liver and right costal margin. Great difficulty was experienced in reducing the last two inches of the intussusception, as the bowel and mesentery were greatly swollen. The distension of the bowel had to be relieved by evacuation of its contents through a cannula inserted about two feet above the intussusception before the bowels could be returned to the abdomen. He rallied well from the operation, but collapsed the same evening, and died about twelve hours after operation. The intussusception was of the ileo-colic variety.

Case 7.—Female infant, aged 7 months. Hydrostatic pressure failed to effect reduction. Laparotomy was done immediately, and the intussusception reduced with difficulty. Child never rallied.

Case 8.—Male infant, aged 6 months. Symptoms for forty-eight hours. Bloody stools were passed. Tumour in left iliac fossa. Hydrostatic pressure failed to reduce the last portion of the intussusception. A $1\frac{1}{2}$ inch incision was made in the right side of the abdomen, through which the tumour was brought out. It was ileo-colic, about one inch from the ileo-cæcal valve. Readily reduced. The infant apparently made a good recovery and remained well for fifteen days, when it was attacked with diarrhœa

and died nineteen days after the operation. No return of intussusception. It being August, diarrhœa was epidemic. The operation appears to have been completely successful.

There can be little doubt that hydrostatic pressure properly applied will effect reduction in a large proportion of cases, and in nearly every case will reduce all but the last portion of the invagination, rendering subsequent operative procedure much simpler. If there is doubt (as often must be the case) as to the completeness of reduction, an incision $1\frac{1}{2}$ inches in length over the ileo-cæcal valve will enable the tumour in most cases to be reached by the finger and hooked out of the abdomen, without exposing a large area of the intestines, as must be done when primary laparotomy is performed. The resulting shock is much less, which is an extremely important matter in operating on young children.

Method of applying hydrostatic pressure.—This is done with a funnel and tube. Normal saline at a temperature of 105° F. is used, the funnel being kept at a height of three feet. The child is fully anæsthetised and the buttock well raised. No attention is paid to the amount of fluid injected, the constant pressure being the one important factor. This is kept up without intermission for half an hour. It is usually not possible to recognise the moment when reduction takes place. The writer believes that the time element is a very important factor, and that injection often fails because not sufficiently systematically applied, and not continued long enough. The only certain sign of reduction having occurred is the presence of a considerable amount of fæcal matter in the water which is ejected after the nozzle is removed from the rectum.

One must allow that the great disadvantage attending non-operative methods of treatment is the uncertainty that remains in the mind of the surgeon as to the completeness of reduction. This uncertainty may deter him from operating until the favourable time is past. This, however,

cannot be urged as an argument in favour of immediate laparotomy, unless it can be shown that the mortality following the latter course of treatment is less than that occurring in cases treated by hydrostatic pressure.

Statistics.—At the discussion on the subject of intussusception at the Cheltenham meeting of the British Medical Association (1901) valuable statistics were brought forward. The general opinion of the speakers was strongly in favour of immediate operation. A careful study of the statistics brought forward hardly bears out this view. Take those of St. Thomas's Hospital,* most valuable because complete, and dealing with consecutive cases from 1875 to 1900.

Of children under 2 years of age, twenty-four were treated without operation. Thirteen recovered, eleven died. Mortality 46 per cent. Eight cases were treated by operation after failure of injection, of whom two recovered. The percentage of cases cured by non-operative measures is 40·5.

Fifty-two cases were treated by immediate laparotomy, of whom fourteen recovered and thirty-eight died—only 27 per cent. of recoveries.

From the year 1897 onward immediate laparotomy was adopted as the routine procedure. Of the thirty-nine cases so treated fifteen recovered and twenty-four died—30·6 per cent. of recoveries, or 10 per cent. less than that obtained by the discarded methods of injection, insufflation, etc.

These statistics are the most complete the writer has been able to find, and they point strongly to the conclusion that in young children, at any rate, the risk is less from non-operative methods than from primary laparotomy. It seems probable to him that the employment of hydrostatic pressure in the manner described above would give better results than the various methods of enemata, injection of air or taxis on which the St. Thomas's Hospital statistics are based.

* 'British Medical Journal,' September the 7th, 1901.

To sum up, the writer believes that, in infants under 2 years, at all events, the method of hydrostatic pressure is effectual in a large proportion of cases, and that the mortality in cases so treated is less than in those treated by immediate laparotomy. Where doubt exists as to the completeness of reduction, this method should be combined with a small incision in the right iliac region through which the ileo-cæcal part of the bowel may be felt, and reduction of a small residual intussusception performed if such is found to exist.

(*Read at the Provincial Meeting, Sheffield, July the* 17*th*, 1905.)

99. TWO CASES OF MULTIPLE XANTHOMA TUBEROSUM.

By C. N. GWYNNE, M.Ch.

The photographs exhibited are from two brothers. They both suffered from the same affection, and the characters and the distribution of the disease were almost identical.

The eldest boy, aged 10 years, was shown by me at a meeting of the Sheffield Medico-Chirurgical Society about seven years ago. He was dark-complexioned, inclined to be delicate, and though well grown, was hardly robust. He had a heart murmur, but was considered by his parents to be in fair health. He returned home after the meeting, ate his supper, and next morning early he died suddenly. No post mortem could be obtained.

The younger brother is now under my care. He showed symptoms of heart-weakness, and found the walking to and from school rather too much for him. He has a mitral systolic murmur. The heart sounds are weak and the extremities cold. The family history points to heart trouble. The mother has heart-disease, and the father has a poor circulation and is easily tired.

The first patches were noticed when the boy was 2½ years old, on the heels and buttocks. On the latter situation one

Fig. 17.

was close to the crease between the buttock and leg and the other on the prominence of the buttock (*vide* Fig. 17). A year previous to the first appearance of these patches he

had an attack of jaundice. When 7 years old the elbows became affected, the patches being quite symmetrical and about the size of a shilling. Since that time the disease has principally attacked his hands. The patch on the left buttock has disappeared. The patches on the elbows which in 1902 were about the size of a shilling have increased to the size of half-a-crown.

The hands have been latest affected and are the most remarkable. The second finger of each hand at and

Fig. 18.

beyond the first phalangeal joint is the site of a remarkable swelling facing the first finger by which the breadth of the joint is increased to nearly double. The colour of the skin is not affected. The patches here are about an inch long and have a firm doughy feel. The articular end of the phalanx is widened out and apparently increased in size. Other fingers are affected to a less degree. Patches of nodules occupy the knuckles, and others are distributed on the extensor tendons on the metacarpus. The elbow patches have the usual dull yellow appearance and so have those placed on the tendo achilles. There is no inconveni-

ence felt from their presence beyond the difficulty of putting on gloves. Those on the tendo achilles cause great inconvenience and affect his walking unless boots are made to accommodate the tumours. They are about the size of a small apple. There are some points in these remarkable cases worthy of note :

(1) The family prevalence. In this connection I may

FIG. 19.

mention that the mother is very dark, and round the eyes the skin is dark-hued.

(2) The co-existence of heart-disease in both cases. This also was the case in the boy reported by A. Pönsgen in 'Virchow's Archives' of February, 1883. A sister aged 15 years had also a well-marked mitral murmur.

(3) There was no history of migraine nor was there any diabetes.

The *etiology* in these cases is involved in obscurity.

The pathology.—The view expressed by Radcliffe-Crocker is that it is a connective-tissue neoplasm situated in the papillary layer of the corion and pushing up the rete into a nodule. He considers inflammation as the primary feature and the whole process of toxæmic origin.

(*Read at the Provincial Meeting, Sheffield, June the* 17*th*, 1905.)

Discussion.

Mr. Jackson Clarke said he recalled that many years ago when assisting Mr. Malcolm Morris to investigate some cases of xanthoma diabeticorum, and in connection with them some cases of xanthoma in general, what struck him about the ordinary xanthoma tumours was that in well-preserved specimens fixed with osmic acid the appearance of xanthelasmic skin resembled that of commencing atheroma of the arteries—*i. e.* previous to calcareous degeneration of the atheromatous thickening. So he thought that the connection between xanthoma and heart disease might be that in some people who had atheroma of the arteries the skin took on a change similar to the early stage of atheroma. That was the impression left on his mind by the investigations.

Dr. C. N. Gywnne (Sheffield), in reply, said that Mr. Jackson Clarke's theory was very extraordinary, but there might be some truth in it. However he could not give any opinion upon it.

INDEX.

PAGE

ABDOMEN, muscles of, involvement of, in infantile paralysis (A. Edmunds) 1, 15
——— ——— *Discussion* (G. A. Sutherland) . . 18
Alliott (A. J.), sudden and unexpected death in children . 117
ALOPECIA, a case of generalised (G. Pernet) . 230, 231
——— ——— ——— *Discussion* (George Carpenter) . . 231
——— universalis, case of (O. Grünbaum) . . . 180
AMAUROTIC idiocy, photograph of a case of (G. Pernet) . 242
ANÆSTHETICS as a cause of sudden death in children (J. Blumfeld) 110
——— deaths under 130, 132
AORTA, disease of (L. Guthrie) 144
——— ——— *Discussion* (E. Cautley) . . . 146
——— ——— ——— (W. Ewart) 145
——— ——— ——— (R. Hutchison) . . . 145
——— ——— ——— (A. Morison) . . . 145
——— ——— ——— (G. A. Sutherland) . . . 146
——— ——— and mitral disease (A. Morison and F. J. Poynton) 148, 226, 229
——— ——— *Discussion* (W. Ewart) . . . 149
——— ——— ——— (F. Fisher) 228
——— ——— ——— (F. J. Poynton) . . . 149
——— ——— ——— (G. A. Sutherland) . . . 229
AORTIC valves, verrucose endocarditis of (E. Cautley) 213, 217
——— ——— *Discussion* (C. W. Chapman) . . 217
——— ——— ——— (W. Ewart) 217
APPENDICITIS in children, points in the diagnosis and treatment of (H. A. Whitelocke) . . . 200, 210
——— ——— *Discussion* (C. W. Chapman) . . . 210
——— ——— ——— (R. C. Dun) . . . 209
——— ——— ——— (P. Lewis) 208
——— ——— ——— (R. C. Lucas) . . . 210
——— ——— ——— (L. Mummery) . . . 208
——— in a hernial sac, report of two cases (H. S. Clogg) 297, 302

PAGE
Appendicitis in a hernial sac. *Discussion* (R. C. Lucas) . 301
——— ——— ——— (C. W. Gwynne) . . . 302
Appetite, perverted 264
Armour (D. J.), case of hypospadias . . . 150
——— discussion on Volkmann's contracture . . 142
Arthritis, chronic, case of (F. J. Poynton) . . 165
——— ——— *Discussion* (E. Hobhouse) . . . 166
——— ——— ——— (F. P. Weber) . . . 166
——— syphilitic, cases of (A. Edmunds and G. A. Sutherland) 170
——— ——— *Discussion* (E. Hobhouse) . . . 172
——— ——— ——— (F. J. Poynton) . . . 172
Ashby (H.), Wightman Lecture: Some of the Neuroses of Early Life 251
Asthma, in infancy 257
Ataxy, cerebellar type of (J. P. Parkinson) . . 62
Athetosis, cerebral diplegic 13
Atrophy, see *Muscular Atrophy.*
Auricle, see *Ear.*

Batchelor (C.), case of exophthalmic goitre in a child aged 3½ years 211, 213
Baths in schools 77
Beach (F.), discussion on case of trichotillomania . . 29
——— ——— cerebral degeneration and fundus changes . 27
——— ——— imbecility and microcephaly . . . 22
Bell's palsy with hemiplegic onset (J. P. Parkinson) . 60
——— ——— *Discussion* (E. Cautley) . . . 61
Bladder, pain in, in appendicitis . . . 207
Blood, examination of, in trophic œdema . . . 6
Blumfeld (J.), anæsthetics as a cause of sudden death in children 110
Bone, rapid softening of, and acute periostitis . . 305
Brain, degeneration of, and fundus changes (S. Stephenson) 25, 28
——— ——— *Discussion* (F. Beach) . . . 27
——— ——— ——— (B. Harman) . . . 27
——— sudden death from (George Carpenter) . . 125
Brickdale (J. M. Fortescue-), see *Fortescue-Brickdale* (J. M.).
Briscoe (J. C.), case of tremor . . . 188, 190
Bromide rash, case of (R. Hutchison) . . . 3
——— ——— *Discussion* (F. J. Poynton) . . . 4

Campbell (H.), case of progressive palsy of cerebral origin . 66
Carpenter (George), case of congenital deformity of the hands: many members of the family show similar and other malformations of the hands 240

PAGE
Carpenter (George) case of congenital morbus cordis . . 147
——— cases of sudden and unexpected death in children . 126
——— discussion on a case of generalised alopecia . . 231
——— discussion on bilateral periostitis of the femora, probably syphilitic 234
——— discussion on fatal result after inserting a needle for supposed empyema 284
——— herpes zoster extending to the finger-tips . . 30
——— specimen of congenital morbus cordis . . 48
——— and Mummery (L.), case of congenital hypertrophic stenosis of the pylorus . . . 242, 247, 249
——— and Stephenson (S.), case of tubercle of the choroid presenting certain clinical and pathological peculiarities . 273
Cases, clinical, shown at the Children's Hospital, Sheffield . 268
Cautley (E.), bilateral periostitis of the femora, probably syphilitic 232, 235
——— congenital heart-disease 19
——— discussion on a case of aortic disease . . 146
——— discussion on congenital hypertrophic stenosis of the pylorus . . . . . 245—247
——— discussion on stricture of the œsophagus . . 224
——— discussion on muscular atrophy, probably of the so-called "peroneal type" 239
——— discussion on spastic diplegia with optic atrophy . 176
——— discussion on congenital morbus cordis . . 51
——— discussion on fatal result after inserting a needle for supposed empyema 285
——— discussion on sudden and unexpected death in children 138
——— imbecility with microcephaly, spasticity, and irregular tremors 21
——— osteomyelitis of the upper end of the femur . . 167
——— verrucose endocarditis of the aortic valves . 213, 217
Cavities, emptying and washing out, fatal result from . 100
Cephalhæmatoma neonatorum, occipital, two cases of (J. H. Evans) 55
——— ——— *Discussion* (R. Jones) . . . 56
Cervical glands, tuberculosis of, and of the lungs and ears (G. F. Hett) 185, 188
——— ——— *Discussion* (J. M. Fortescue-Brickdale) . 188
——— ——— ——— (L. Guthrie) . . . 188
——— ——— ——— (G. A. Sutherland) . . . 187

PAGE
Chapman (C. W.), discussion on a case of erythema multiforme following vaccination 155
——— discussion on some points in the diagnosis and treatment of appendicitis in children 210
——— notes on a case of hysteria in a girl aged 10½ years 193, 197
Chest, exploratory puncture of, death from (George Carpenter) 285
Chloroform poisoning and sudden death . . 110, 130, 132
Choroid, tubercle of, case presenting certain clinical and pathological peculiarities (George Carpenter and S. Stephenson) 273
Clarke (E.), case of double coloboma of the iris . . 231
——— discussion on silver deposit in cornea . . 226
Clarke (J. J.), congenital dislocation of the hip-joint, some practical points in Lorenz's manipulative operation and the after-treatment 290, 294
——— discussion on a case of late rickets . . . 182
——— discussion on two cases of multiple xanthoma tuberosum 322
Cleft-palate, ill-developed upper lateral incisor tooth as a forerunner of (R. C. Lucas) 31
Clogg (H. S.), appendicitis in a hernial sac: a report of two cases 297, 302
Club-foot, acquired, with marked hereditary history (D. Drew) 172
Coloboma, double, of the iris (E. Clarke) 231
Congenital absence of gall-bladder and bile-ducts (E. Hobhouse) 177
——— atresia, the tricuspid valve (B. M. H. Rogers and J. M. Fortescue-Brickdale) 46
—— bilateral and symmetrical dislocation of the crystalline lenses (N. B. Harman) 23
—— deformity of both forearms (R. C. Dun) . . 198
—— ——— *Discussion* (R. C. Lucas) . . . 199
——— deformity of the hands, etc., case of (George Carpenter) 240
——— diaphragmatic hernia, two cases (S. Gilford) . . 38
——— dislocation of the hip-joint: some practical points in Lorenz's method (J. J. Clarke) . . 290, 294
—— ——— *Discussion* (A. H. Tubby) . . . 294
—— dislocation of the hip, unilateral, three cases of (D. Drew) 57
——— ——— *Discussion* (R. Jones) . . . 58
— ——— ——— (L. McGavin) . . . 57
—— heart-disease (George Carpenter) . . 48, 51, 147
—— ——— *Discussion* (E. Cautley) . . 19, 51

PAGE
Congenital heart disease, *Discussion* (S. Curl) . . 50
—— hypertrophic stenosis of the pylorus (George Carpenter and P. L. Mummery) . . . 242, 247, 249
—— —— *Discussion* (E. Cautley) . . 245—247
—— —— —— (F. J. Poynton) . . . 246
—— —— —— (F. Fisher) 247
—— —— —— (A. Morison) . . . 247
—— morbus cordis, specimen of (George Carpenter) 48, 51
—— —— *Discussion* (E. Cautley) . . . 51
—— —— —— (S. Curl) 50
—— occlusions of the small intestine (J. G. Emanuel) . 155
—— stricture of the œsophagus (R. C. Dun) . 218, 224
—— —— *Discussion* (R. C. Lucas) . . . 223
—— —— —— (T. R. Whipham) . . . 224
—— —— —— (E. Cautley) . . . 224
—— trophic œdema (O. Grünbaum . . . 4, 13
—— —— *Discussion* (C. O. Hawthorne) . . 12
—— —— —— (P. Parkinson) . . . 12
—— —— —— —— (G. Pernet). . . . 12
—— —— —— —— (A. H. Tubby) . . . 10
—— —— —— —— (F. P. Weber) . . . 11
—— unilateral paresis of the third and fourth cranial nerves (N. B. Harman) 158, 162
—— —— *Discussion* (J. P. Parkinson). . . 160
—— —— —— (S. Stephenson) . . . 161
—— unilateral ptosis (E. Pritchard) . . . 152
—— —— *Discussion* (C. O. Hawthorne) . . 153
Convulsions and sudden death 86
Cornea, injury to, following instrumental delivery (S. Stephenson) 65
—— silver deposit in (S. Stephenson) . . . 225
—— —— —— *Discussion* (E. Clarke) . . . 226
Cranial nerves, third and fourth, case of unilateral congenital paresis of (N. B. Harman) . . . 158, 162
—— *Discussion* (J. P. Parkinson) . . . 160
—— —— (S. Stephenson) 161
Crystalline lens, congenital and symmetrical dislocation of (N. B. Harman) 23
Curl (S.), specimen of congenital morbus cordis . . 50
—— —— tuberculous ulceration of the intestine and tuberculosis of the right Fallopian tube. . . 70
Cyst, see *Dermoid*.

PAGE
DEATH, sudden and unexpected in children (A. J. Alliott, George Carpenter, J. M. Fortescue-Brickdale) . 117—136
——— ——— *Discussion* (E. Cautley) . . . 138
——— ——— ——— (A. Edmunds) . . . 137
——— ——— ——— (G. Pernet) 137
——— ——— ——— (F. Taylor) 139
——— ——— after insertion of needle for supposed empyema (J. H. Wilks) 279, 286
——— ——— *Discussion* (George Carpenter) . . 284
——— ——— ——— (E. Cautley) . . . 285
——— ——— ——— (R. C. Lucas) . . . 284
——— ——— ——— (G. A. Sutherland) . . . 283
——— ——— anæsthetics as a cause of (J. Blumfeld) . 110
——— ——— functional causes of (J. P. Parkinson) . 85
——— ——— organic causes of (C. Macalister) . . 72
——— ——— status lymphaticus, and (J. W. T. Walker) . 90
——— ——— surgical causes of (G. H. Tubby) . . 94
——— ——— undetermined causes of (George Carpenter) . 132
DEGLUTITION in new-born infants. . . . 255
DERMOID cyst of the auricle (P. L. Mummery) . . 18
DIARRHŒA in infantile appendicitis . . 205, 209
DIPHTHERIA, heart failure after 89
DIPLEGIA, spastic with optic atrophy (E. Pritchard) . 174, 177
——— *Discussion* (E. Cautley) 176
——— ——— (E. Hobhouse) 175
——— ——— (G. A. Sutherland) 176
DIRT-EATING in early life 263
Drew (D.), acquired club-foot with marked hereditary history . 172
——— case of old-standing dislocation of the head of the radius 59
——— discussion on osteomyelitis of the upper end of the femur 168
——— excision of both hip-joints for tuberculous disease . 173
——— three cases of unilateral congenital dislocation of the hip 57
Dun (R. C.), case of congenital deformity of both forearms . 198
——— ——— stricture of the œsophagus . . 218, 224
——— ——— median hare-lip 294
——— discussion on some points in the diagnosis and treatment of appendicitis in children 209
EAR, dermoid cyst of (P. L. Mummery) . . . 18
——— tuberculosis of, and of the lungs and cervical glands (G. F. Hett) 185, 188

PAGE
Ear, tuberculosis of, and of the lungs and cervical glands. *Discussion* (J. M. Fortescue-Brickdale) . . . 188
——— ——— ——— (L. Guthrie) . . . 188
——— ——— ——— (G. A. Sutherland) . . . 187
Edmunds (A.), case of syphilitic arthritis . . . 170
——— discussion on hypertrophy of right lower limb . 1
——— ——— on sudden and unexpected death in children . 137
——— infantile paralysis with permanent involvement of the abdominal muscles 15
Emanuel (J. G.), multiple congenital occlusions of the small intestine 155
Embolism and sudden death 98
Emotions and sudden death 88
Empyema, supposed, death after inserting a needle for (J. H. Wilks) 279, 286
——— ——— *Discussion* (G. A. Sutherland) . . 283
——— ——— ——— (R. C. Lucas) . . . 284
——— ——— ——— (George Carpenter) . . . 284
——— ——— ——— (E. Cautley) . . . 285
Endocarditis, verrucose, of the aortic valves (E. Cautley) 213, 217
——— ——— *Discussion* (C. W. Chapman) . . 217
——— ——— ——— (W. Ewart) 217
Enuresis, cause of 256
Environment, influence of 266
Eruptions, too rapid healing of 137
Erythema multiforme following vaccination (G. Pernet) . 155
——— ——— *Discussion* (C. W. Chapman) . . . 155
Evans (J. H.), two cases of occipital cephalhæmatoma neonatorum 55
Ewart (W.), discussion on a case of aortic disease . . 145
——— discussion on a case of aortic and mitral disease . 149
——— discussion on verrucose endocarditis of the aortic valves 217
——— discussion on Volkmann's contracture . . 141
Extremity, lower, hypertrophy of (C. O. Hawthorne) . 1
——— ——— *Discussion* (A. Edmunds) . . . 1
——— ——— ulcer of 1
——— upper, congenital deformity of both forearms (R. C. Dun) 198
——— ——— *Discussion* (R. C. Lucas) . . . 199
Eye, see *Choroid*, *Cornea*, *Fundus*, and *Iris*.

Fallopian tube, tuberculosis of (S. Curl) . . . 70
Faradism in muscular atrophy 237

PAGE

FEMUR, bilateral periostitis of, probably syphilitic (E. Cautley) 232, 235
——— ——— *Discussion* (H. Skelding) . . . 233
——— ——— ——— (F. J. Poynton) . . . 233
——— ——— ——— (G. A. Sutherland) . . . 234
——— ——— ——— (George Carpenter) . . . 234
——— osteomyelitis of upper end of (E. Cautley) . . 167
——— ——— *Discussion* (D. Drew) . . . 168
FEVER recurring in infancy 258
——— septic, sudden death in 80
Fisher (T.), discussion on cases of combined aortic and mitral disease 228
FOREARM, see *Extremity, Upper.*
FOREIGN body in the œsophagus (A. T. Marshall) . . 157
——— bodies, sudden death and 107
Fortescue-Brickdale (J. M.), discussion on a case of tuberculosis of the lungs, the cervical glands, and the ears . 188
——— ——— sudden and unexpected death in children . 118
——— see also Rogers (B. M. H.) and Fortescue-Brickdale (J. M.).
FUNDUS changes and cerebral degeneration (S. Stephenson) 25, 28
——— *Discussion* (F. Beach) 27
——— ——— (N. B. Harman) 27

GALL-BLADDER and bile-ducts, congenital absence of (E. Hobhouse) 177
GENERAL paralysis, case of juvenile (J. Taylor) . 52, 55
——— ——— *Discussion* (F. P. Weber) . . . 54
Gilford (S.), two cases of congenital diaphragmatic hernia . 38
GOITRE, exophthalmic, in a child aged 3½ years (C. Batchelor) 211, 213
——— ——— *Discussion* (R. C. Lucas) . . . 212
——— ——— ——— (S. Stephenson) . . . 212
——— ——— ——— (G. A. Sutherland) . . . 213
——— ——— in a boy aged 12 years . . . 46
——— ——— *Discussion* (G. A. Sutherland) . . 65
Grünbaum (O.), case of alopecia universalis . . . 180
——— congenital trophic œdema 4
GUMMATA, multiple (J. P. Parkinson) . . . 249
Guthrie (L.), case of aortic disease . . 144, 147
——— discussion on a case of late rickets . . . 181
——— discussion on a case of tremor . . . 189

PAGE

Guthrie (L.), discussion on a case of tuberculosis of the lungs, the cervical glands, and the ears 188
——— discussion on sudden death in children . . 135
——— discussion on Volkmann's contracture . . 142
——— two cases of polio-encephalitis associated with measles . 13
Gwynne (C. N.), case of osteomyelitis with suppurative pericarditis treated by incision and drainage; recovery 269, 272
——— discussion on appendicitis in a hernial sac . . 301
——— two cases of multiple Xanthoma tuberosum . 318, 322

Habit spasm (R. Hutchison) 2, 3
——— ——— *Discussion* (C. H. Milburn) . . . 3
——— ——— ——— (P. Parkinson) . . . 2
——— ——— ——— (G. A. Sutherland) . . . 2
Hæmophilia, hereditary in the male line (E. Pritchard) . 150
——— ——— *Discussion* (P. Lewis) . . . 151
——— and unexpected death. 96
Hæmorrhages into the supra-renal bodies . . 80
——— spontaneous 97
Hands, congenital deformity of, etc., case of (George Carpenter) 240
Hair, pulling out, mania for 29
Hare-lip, ill-developed upper lateral incisor tooth as forerunner of (R. C. Lucas) 31
——— median, case of (R. C. Dun) . . . 294
——— ——— *Discussion* (R. C. Lucas) . . . 297
Harman (N. B.), case of congenital bilateral and symmetrical dislocation of the crystalline lenses . . . 23
——— case of unilateral congenital paresis of third and fourth cranial nerves 158, 162
——— discussion on cerebral degeneration and fundus changes 27
Hawthorne (C. C.), discussion on a case of congenital unilateral ptosis 153
——— discussion on case of congenital trophic œdema . 12
——— hypertrophy affecting the right lower limb . . 1
Head-nodding, cause of 257
Heart, congenital malformation of . . . 47
——— ——— death from (George Carpenter) . . 120
——— disease, congenital (E. Cautley) . . . 19
——— ——— congenital, case of (George Carpenter) . 147
——— ——— congenital, specimen of (George Carpenter) 48—51
——— ——— *Discussion* (E. Cautley and S. Curl) . . 51
——— ——— and sudden death 83

PAGE

HEART-DISEASE, see also *Mitral Disease; Tricuspid Valve.*
HEMIPLEGIC spasticity and inco-ordination in a child aged 5 years (E. Hobhouse) . . . 169, 170
——— ——— *Discussion* (F. J. Poynton) . . . 170
——— ——— ——— (F. P. Weber) . . . 170
HERNIA, congenital diaphragmatic, two cases (S. Gilford) . 38
HERNIAL sac, appendicitis in, report of two cases (H. S. Clogg) 297, 302
——— ——— *Discussion* (R. C. Lucas) . . . 301
——— ——— ——— (C. W. Gwynne) . . . 301
HERPES zoster extending to the finger-tips (George Carpenter) 30
HEREDITARY hæmophilia in the male line (E. Pritchard) . 150
——— ——— *Discussion* (P. Lewis) . . . 151
——— history in acquired club-foot (D. Drew) . . 172
——— œdema 9
Hett (G. F.), case of tuberculosis of the lungs, the cervical glands, and the ears 185, 188
HIP-JOINT, congenital dislocation of, some practical points in Lorenz's method (J. J. Clarke) . . 290, 294
——— ——— *Discussion* (A. H. Tubby) . . . 294
——— dislocation of, unilateral congenital, three cases of (D. Drew) 57
——— ——— *Discussion* (R. Jones) . . . 58
——— ——— ——— (L. McGavin) . . . 57
——— excision of both, for tuberculous disease (D. Drew) . 172
Hobhouse (E.), congenital absence of gall-bladder and bile-ducts 177
——— discussion on a case of chronic arthritis . . 166
——— discussion on a case of spastic diplegia with optic atrophy 175
——— discussion on a case of syphilitic arthritis . . 172
——— hemiplegic spasticity and inco-ordination in a child aged 5 years 169
——— interstitial nephritis in a child aged 6½ years, with renal retinitis 177
Hutchison (R.), case of bromide rash . . . 3
——— case of habit spasm 2
——— case of late rickets . . . 181, 183
——— discussion on a case of aortic disease . . 145
HYPNOTISM in habit spasm 3
HYPOSPADIAS, case of (D. J. Armour) . . . 150
——— ——— *Discussion* (P. Lewis) . . . 150

PAGE
Hysteria in a girl aged 10½ years (C. W. Chapman). 193, 197
——— ——— *Discussion* (R. C. Lucas) . . . 197
——— ——— ——— (G. E. Shuttleworth) . . 197

Idiocy, see *Amaurotic Idiocy, Imbecility.*
Imbecility with microcephaly, spasticity, and irregular tremors (E. Cautley) 121
——— ——— *Discussion* (F. Beach) . . . 22
——— ——— ——— (P. Parkinson) . . . 22
Intestine, small, multiple congenital occlusions of (J. G. Emanuel) 155
——— tuberculous ulceration of (S. Curl) . . . 70
Intussusception in infants, treatment of (G. Wilkinson) . 313
Iris, double coloboma of (E. Clarke) . . . 231
Ischæmic paralysis, see *Volkmann's Contracture.*

Jaffrey (F.), two cases of Volkmann's contracture . . 140
Jaundice in a child, with chronic enlargement of the spleen (F. P. Weber) 191, 193
— —— ——— *Discussion* (G. A. Sutherland) . . 192
Jaws, necrosis of the alveolar margins of . . . 305
Joint-disease, case of Still's type of (F. P. Weber) . . 162
Jones (R.), discussion on cephalhæmatoma neonatorum . 56
——— discussion on dislocation of the head of the radius . 59
——— discussion on unilateral congenital dislocation of the hip 58

Kelson (W. H.), fibroma and lupus of the larynx . . 143
Kerato-malacia, case of (S. Stephenson) . . . 184
Kidney, see *Nephritis.*

Labour, difficult 65
Lacrymal sac, tuberculosis of (S. Stephenson) . . 184
Laryngismus stridulus and sudden death . . . 87
Larynx, fibroma and lupus of (W. H. Kelson) . . 143
——— papillomata of (L. Marshall) . . . 250
Leader (H.), notes on a case of lympho-sarcoma . . 286
Lewis (P.), discussion on a case of hæmophilia hereditary in the male line 151
——— discussion on a case of hypospadias . . . 150
——— discussion on some points in the diagnosis and treatment of appendicitis in children . . . 208
——— discussion on Volkmann's contracture . . 142

PAGE
LICHEN planus, case of (G. Pernet) . . . 153
LIMBS, see *Extremities*.
LORENZ'S manipulative operation and the after-treatment . 290
Lucas (R. C.), discussion on appendicitis in a hernial sac . 301
——— discussion on a case of congenital deformity of both forearms 199
——— discussion on a case of exophthalmic goitre in a child aged 3½ years 212
——— discussion on a case of hysteria in a girl aged 10½ years 197
——— discussion on fatal result after inserting a needle for supposed empyema 284
——— discussion on some points in the diagnosis and treatment of appendicitis in children . . . 210
——— on an ill-developed upper incisor tooth as a forerunner of harelip or cleft palate 31
LUNGS, collapse of, and sudden death in . . . 84
——— sudden death from (George Carpenter) . . 123
——— tuberculosis of, and of the cervical glands and ears (G. F. Hett) 185, 188
——— ——— *Discussion* (J. M. Fortescue-Brickdale) . 188
——— ——— ——— (L. Guthrie) . . . 188
——— ——— ——— (G. A. Sutherland) . . . 187
LUPUS of the larynx (W. H. Kelson) . . . 143
LYMPHO-SARCOMA, notes on a case of (H. Leader) . . 286

Macalister (C.), organic causes of sudden and unexpected deaths in children 72
McGavin (L.), discussion on unilateral congenital dislocation of the hip 57
MARASMUS and sudden death 86
Marshall (A. T.), collar-stud impacted in the œsophagus and perforating the trachea 156
Marshall (L.), case of laryngeal papillomata—death from asphxyia 250
MASTURBATION in early life 262
MEASLES, associated with polio-encephalitis (L. Guthrie) . 13
——— and muscular atrophy 240
MICROCEPHALY with imbecility, spasticity and irregular tremors (E. Cautley) 21
——— ——— *Discussion* (F. Beach) . . . 22
——— ——— ——— (P. Parkinson) . . . 22
Milburn (C. H.), discussion on habit spasm . . . 3

PAGE

MITRAL and aortic disease (A. Morison) . . . 148
——— ——— *Discussion* (W. Ewart) . . . 149
——— ——— ——— (F. J. Poynton) . . . 149
——— ——— cases of combined (F. J. Poynton) . 226, 229
——— ——— ——— (T. Fisher) . . . 228
——— ——— ——— *Discussion* (G. A. Sutherland) . 229
Morison (A.), case of aortic and mitral disease . . 148
——— discussion on a case of aortic disease . . 145
——— discussion on a case of congenital hypertrophic stenosis of the pylorus 247
MORTALITY, infantile, causes of 72, 85, 90, 94, 110, 117, 120, 136
Mummery (P. L.), dermoid cyst of the auricle . . 18
——— discussion on a case of late rickets . . . 183
——— discussion on some points in the diagnosis and treatment of appendicitis in children . . . 208
MURMURS, cardiac 229
MUSCULAR atrophy, probably so-called "peroneal type" (F. P. Weber) 236, 240

NEPHRITIS, interstitial, in a child aged 6½ years, with renal retinitis (E. Hobhouse) 177
NERVES, see *Cranial Nerves*.
NERVOUS system, influence of, in sudden death . . 108
NEUROSES of early life. (Wightman Lecture.) (H. Ashby) . 251
NIGHT terrors in infancy 259
NYSTAGMUS in a case of tremor 190

ŒDEMA, acute and sudden death 105
——— congenital trophic (O. Grünbaum) . . . 4, 13
——— ——— *Discussion* (E. C. Hawthorne) . . 12
——— ——— ——— (P. Parkinson) . . . 12
——— ——— ——— (G. Pernet) . . . 12
——— ——— ——— (A. H. Tubby) . . . 10
——— ——— ——— (F. P. Weber) . . . 11
ŒSOPHAGUS, collar-stud impacted in, perforating the trachea (A. T. Marshall) 157
——— congenital stricture of (R. C. Dun) . . 218, 224
——— ——— (E. Cautley) 224
——— ——— *Discussion* (R. C. Lucas) . . . 223
——— ——— ——— (T. R. Whipham) . . . 224
OPERATIONS, surgical, death following (George Carpenter) . 128
OPIUM, use of, in abdominal surgery . . . 211

PAGE
Optic atrophy in case of spastic diplegia (E. Pritchard) 174, 177
——— ——— *Discussion* (E. Cautley) . . . 176
——— ——— ——— (E. Hobhouse) . . . 175
——— ——— ——— (G. A. Sutherland) . . . 176
Osteomyelitis and periostitis of the spine (A. H. Tubby) . 302
——— with suppurative pericarditis treated by incision and drainage (C. N. Gwynne) . . . 269, 272
——— ——— *Discussion* (H. B. Robinson) . . 272
——— of upper end of the femur (E. Cautley) . . 167
——— ——— *Discussion* (D. Drew) . . . 168
Overlying as a cause of death 87

Paralysis, birth, case of (J. P. Parkinson) . . 250
—— infantile, with permanent involvement of abdominal muscles (A. Edmunds) 15
——— ——— *Discussion* (G. A. Sutherland) . . 18
——— progressive, of cerebral origin (H. Campbell) . . 66
——— see also *General paralysis.*
Parkinson (J. P.), case of ataxy of the cerebellar type . 62
——— case of Bell's palsy with hemiplegic onset . . 60
——— case of birth paralysis 250
——— discussion on a case of unilateral congenital paresis of third and fourth cranial nerves . . . 160
——— discussion on congenital trophic œdema . . 12
——— discussion on habit spasm 2
——— discussion on imbecility and microcephaly . . 22
——— functional causes of sudden and unexpected death in children 85
——— multiple gummata 249
Pathological specimens shown at the Children's Hospital, Sheffield 268
Pericarditis, suppurative, with osteomyelitis, treated by incision and drainage (C. N. Gwynne) . . 269, 272
——— ——— *Discussion* (H. B. Robinson) . . 272
Periostitis and osteomyelitis of the spine (A. H. Tubby) . 302
— probably syphilitic, of the femora (E. Cautley) 232, 235
——— ——— *Discussion* (George Carpenter) . . 234
——— ——— ——— (F. J. Poynton) . . . 233
——— ——— ——— (H. Skelding) . . . 233
——— ——— ——— (G. A. Sutherland) . . . 234
Pernet (G.), case of erythema multiforme following vaccination 155
——— case of generalised alopecia . . . 230

PAGE
Pernet, G., case of lichen planus 153
——— case of trichotillomania 28
——— discussion on a case of alopecia universalis . . 180
——— discussion on congenital trophic œdema . . 12
——— discussion on sudden and unexpected death in children 137
——— photograph of a case of amaurotic idiocy . . 242
Phthisis in a girl aged 12 years 185
——— see also *Lungs*.
Pneumonia, double and acute periostitis, etc. . . 305
Polio-encephalitis, associated with measles (L. Guthrie) . 13
Poliomyelitis in paresis of cranial nerves . . 159, 161
Poynton (F. J.), case of chronic arthritis . . . 165
——— cases of combined aortic and mitral disease . 226, 229
——— discussion on bilateral periostitis of the femora, probably syphilitic 233
——— discussion on bromide rash . . . 4
——— discussion on a case of aortic and mitral disease . 149
——— discussion on a case of congenital hypertrophic stenosis of the pylorus 246
——— discussion on a case of syphilitic arthritis . . 172
——— discussion on hemiplegic spasticity and inco-ordination in a child aged 5 years 170
Pritchard (E.), case of congenital unilateral ptosis . . 152
——— case of hæmophilia hereditary in the male line . 150
——— case of spastic diplegia with optic atrophy . . 174
Ptosis, congenital unilateral (E. Pritchard) . . 152
——— ——— *Discussion* (C. O. Hawthorne) . . 153
Pyæmia and sudden death 106
Pyloroplasty, remarks on . . . 246, 249
Pylorus, hypertrophic stenosis of, congenital (George Carpenter and P. L. Mummery) . . . 242, 247, 249
——— ——— *Discussion* (E. Cautley) . . 245—247
——— ——— ——— (T. Fisher) . . . 247
——— ——— ——— (A. Morison) . . . 247
——— ——— ——— (F. J. Poynton) . . . 246

Radius, old-standing dislocation of head of (D. Drew) . 59
——— ——— *Discussion* (R. Jones) . . 59
Rash, see *Bromide rash*.
Regurgitation, aortic 229
Retinitis, renal, in interstitial nephritis (E. Hobhouse) . 177
Rickets, late, case of (R. Hutchison) . . . 181

PAGE

Robinson (H. B.), discussion on a case of osteomyelitis with suppurative pericarditis treated by incision and drainage: recovery 272
Rogers (B. M. H.) and Fortescue-Brickdale (J. M.), congenital atresia of the tricuspid valve 46

Sarcoma, see *Lympho-sarcoma.*
Sheffield children's hospital, cases and specimens shown at . 268
Shock, surgical, and sudden death 99
Shuttleworth (G. E.), discussion on a case of hysteria in a girl aged 10½ years 197
Silver deposit in cornea (S. Stephenson) . . . 225
——— ——— *Discussion* (E. Clarke) 226
Skelding (H.), discussion on bilateral periostitis of the femora, probably syphilitic 233
Smallpox, sudden death in 82
Spasm, habit, case of (R. Hutchison) . . . 23
——— ——— *Discussion* (C. H. Milburn) . . . 3
——— ——— ——— (P. Parkinson) 2
——— ——— ——— (G. A. Sutherland) . . . 2
Specimens, see *Pathological specimens.*
Spine, acute osteomyelitis and periostitis of (A. H. Tubby) . 302
——— curvature of 17
Spleen, chronic enlargement of, and jaundice in a child (F. P. Weber) . . . . . 191—193
——— ——— *Discussion* (G. A. Sutherland) . . 192
Status lymphaticus and sudden death . . . 92
——— ——— in children (J. W. T. Walker) . . 92
Stephenson (S.), case of cerebral degeneration with fundus changes 25, 28
——— case of Graves' disease in a boy aged 12 years . 64
——— case of kerato-malacia 184
——— case of tuberculosis of the lacrymal sac . . 184
——— discussion on a case of exophthalmic goitre in a child aged 3½ years 212
——— discussion on a case of tremor . . . 190
——— discussion on a case of unilateral congenital paresis of third and fourth cranial nerves . . . 161
——— injury to the cornea following instrumental delivery . 65
——— silver deposit in cornea 225
Supra-renal bodies, hæmorrhages into . . . 80

PAGE
Sutherland (G. A.), case of syphilitic arthritis . . 171
—— discussion on bilateral periostitis of the femora, probably syphilitic 234
—— discussion on a case of aortic disease . . 229
—— discussion on a case of exophthalmic goitre in a child aged $3\frac{1}{2}$ years 213
—— discussion on a case of Graves' disease . 65
—— discussion on a case of habit spasm . . . 2
—— discussion on a case of late rickets . . . 182
—— discussion on a case of spastic diplegia with optic atrophy 176
—— discussion on case of tremor . . . 190
—— discussion on a case of tuberculosis of the lungs, the cervical glands, and the ears 187
—— discussion on a case of combined aortic and mitral disease 229
—— discussion on fatal result after inserting a needle for supposed empyema 283
—— discussion on infantile paralysis . . . 18
—— discussion on jaundice in a child, with chronic enlargement of the spleen 192
Syphilis and congenital heart-disease . . . 51
—— and juvenile general paralysis . . . 52
—— and sudden death 136
—— in periostitis of the femora . . . 235
—— see also *Arthritis*.
Syphilitic (probable) periostitis of the femora (E. Cautley) 232, 235
—— —— *Discussion* (George Carpenter) . . 234
—— —— —— (F. J. Poynton) . . . 233
—— —— —— (H. Skelding) . . . 233
—— —— —— (G. A. Sutherland) . . . 234

Taylor (F.), discussion on sudden and unexpected death in children 139
—— (J.), case of juvenile general paralysis . . 53
Testis, undescended, double, case of (A. H. Tubby) . . 71
Thorax, exploratory puncture of, and sudden death . 118, 279
Thrombosis and sudden death 98
Tooth, upper lateral incisor, ill-developed, as a forerunner of hare-lip and cleft palate (R. C. Lucas) . . . 31
Toxic poisoning and sudden death . . . 102
Tremor, case of (J. C. Briscoe) . . . 188, 190
—— —— *Discussion* (L. Guthrie) . . 189

PAGE

Tremor, case of, *Discussion* (S. Stephenson) . . 190
——— ——— ——— (G. A. Sutherland) 190
——— irregular, with imbecility and microcephaly (E. Cautley) 21
——— ——— *Discussion* (F. Beach) . . . 22
——— ——— ——— (P. Parkinson) . . . 22
Trichotillomania, case of (G. Pernet) . . . 28
——— ——— *Discussion* (F. Beach) . . . 29
Tricuspid valve, congenital atresia of (B. M. H. Rogers and J. M. Fortescue-Brickdale) 46
Tubby (A. H.), acute osteomyelitis and periostitis of the spine . 302
——— case of double undescended testis . . . 71
——— discussion on congenital dislocation of the hip-joint . 294
——— discussion on congenital trophic œdema . . 10
——— surgical causes of sudden and unexpected death . 94
Tuberculosis, acute miliary, diagnosis of (George Carpenter) 277
——— of the lungs, cervical glands, and the ears (G. F. Hett) 185, 188
——— ——— *Discussion* (J. M. Fortescue-Brickdale) . 188
——— ——— ——— (L. Guthrie) . . . 188
——— ——— ——— (G. A. Sutherland) . . . 187
——— and sudden death 104
Typhoid fever, sudden death in 81

Ulcer of the leg 1
Uræmic poisoning and sudden death . . . 103

Vaccination, erythema multiforme following (G. Pernet) . 155
——— ——— *Discussion* (C. W. Chapman) . . 155
Varicella, sudden death in 82
Viscera, displacement of 38
Volkmann's contracture, two cases of (F. Jaffrey) . 140, 142
——— ——— *Discussion* (D. Armour) . . . 142
——— ——— ——— (W. Ewart) . . . 141
——— ——— ——— (L. Guthrie) . . . 142
——— ——— ——— (P. Lewis) 141

Walker (J. W. T.), the status lymphaticus and sudden and unexpected death in children 90
Weber (F. Parkes), case of muscular atrophy, probably of the so-called "peroneal type" . . . 236, 240
——— case of Still's type of chronic joint disease in children . 162
——— discussion on a case of chronic arthritis . . 166
——— discussion on a case of late rickets . . . 182
——— discussion on congenital trophic œdema . . 11

PAGE

Weber (F. Parkes), discussion on hemiplegic spasticity and inco-ordination in a child aged 5 years . . . 170
——— discussion on juvenile general paralysis . . 54
——— jaundice in a child, with chronic enlargement of the spleen 191
Whipham (T. R.), discussion on a case of congenital stricture of the œsophagus 224
Whitelocke (H. A.), some points in the diagnosis and treatment of appendicitis in children . . 200, 210
Wightman Lecture: Neuroses of Early Life (H. Ashby) . 251
Wilkinson (G.), treatment of intussusception in infants . 313
Wilks (J. H.), death after inserting a needle for supposed empyema 279, 286

Xanthoma tuberosum, multiple, two cases of (C. N. Gwynne) 318, 322
——— *Discussion* (J. Clarke) . . . 322

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